AGRICULTURAL RESEARCH INSTITUTE

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PROTOZOOLOGY

PROTOZOOLOGY

A MANUAL

FOR MEDICAL MEN, VETERINARIANS
AND ZOOLOGISTS

BY

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FORMERLY PROTOZOOLOGIST TO THE LONDON SCHOOL OF TROPICAL MEDICINE

IN TWO VOLUMES

WITH 565 ILLUSTRATIONS AND 20 COLOURED PLATES

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PART II

SYSTEMATIC DESCRIPTION OF THE PROTOZOA
WITH SPECIAL REFERENCE TO PARASITIC AND COPROZOIC FORMS

(Continued)

CLASSIFICATION.

CLASS: **SPOROZOA**

  SUB-CLASS: **Coccidiomorpha**

    Order: COCCIDIIDA

      Sub-Order: **Eimeriidea**

        Family: SELENOCOCCIDIIDÆ

         ,,   CRYPTOSPORIDIIDÆ

         ,,   EIMERIIDÆ

        Sub-Family: CYCLOSPORINÆ

          ,   ISOSPORINÆ

          ,,   EIMERIINÆ

          ,,   BARROUXIINÆ

          ,,   CARYOSPORINÆ

          ,,   PFEIFFERINELLINÆ

      Family: CARYOTROPHIDÆ

        ,,   AGGREGATIDÆ

        ,,   LANKESTERELLIDÆ

      Sub-Family: SCHELLACKIINÆ

        ,,   LANKESTERELLINÆ

      Sub-Order: **Hæmosporidiidea**

        Family: HÆMOPROTEIDÆ

          Genus: **Hæmoproteus**

            ,,   **Leucocytozoon**

        Family: PLASMODIIDÆ

      Sub-Order: **Piroplasmidea**

        Family: BABESIIDÆ

         ,,   THEILERIIDÆ

Parasites of Doubtful Nature

    Toxoplasma
    Elleipsisoma
    Pirhæmocyton
    Cytamœba

Structures of Doubtful Nature

    Anaplasma
    Grahamella
    Bartonella
    Rickettsia
    Paraplasma
    Microsoma
    Cingula
    Immanoplasma
    Globidiellum
    Hæmotractidium
    Hæmohormidium

Order: ADELEIDA

  Sub-Order: **Adeleidea**

    Family: DOBELLIIDÆ

      ,,   LEGERELLIDÆ

      ,,   ADELEIDÆ

    Sub-Family: ADELEINÆ

      ,,   KLOSSIINÆ

      ,,   CHAGASELLINÆ

    Family: KLOSSIELLIDÆ

    Sub-Order: **Hæmogregarinidea**

      Family: HÆMOGREGARINIDÆ

        ,,   HEPATOZOIDÆ

        ,,   KARYOLYSIDÆ

SUB-CLASS: **Gregarinina**

  Order: SCHIZOGREGARINIDA

    Genus: **Ophryocystis**

      ,,   **Schizocystis**

      ,,   **Caulleryella**

      ,,   **Lipotropha**

      ,,   **Menzbieria**

      ,,   **Porospora**

      ,,   **Spirocystis**

      ,,   **Selenidium**

      ,,   **Merogregarina**

  Order: EUGREGARINIDA

    Sub-Order: **Acephalinidea**

      Family: MONOCYSTIDÆ

        ,,   LANKESTERIIDÆ

    Sub-Order: **Cephalinidea**

      Family: GREGARINIDÆ

        ,,   STENOPHORIDÆ

        ,,   DIDYMOPHYIDÆ

        ,,   DACTYLOPHORIDÆ

        ,,   ACTINOCEPHALIDÆ

        ,,   STYLORHYNCHIDÆ

        ,,   DOLIOCYSTIDÆ

The class Sporozoa includes Protozoa, which are exclusively parasitic in habit, and which live in the cells or body fluids of vertebrate and invertebrate animals. Leuckart (1879), to whom the name Sporozoa is due, included in the class only the coccidia and gregarines, but later observers extended it to embrace other groups. Schaudinn (1900) finally divided the Sporozoa into two sub-classes—the Telosporidia, to include the gregarines, coccidia, and hæmosporidia; and the Neosporidia, to include the Myxosporidiida, Microsporidiida, Actinomyxidiida, and Sarcosporidia. Though the members of both these sub-classes produce resistant spores, those of the former are not homologous with those of the latter, and it is clear that their affinities are not sufficiently close to justify their inclusion in the same class. Hartmann (1907), recognizing this discrepancy, established two classes, for which he employed Schaudinn's names, Neosporidia and Telosporidia. As Leuckart (1879) founded the class Sporozoa to include the coccidia and gregarines, which are the members of Hartmann's class Telosporidia, there seems to be no reason why Leuckart's name should not be retained. Accordingly, the class Sporozoa will be regarded as including the coccidia and gregarines, together with the blood-inhabiting hæmosporidia and hæmogregarines, which are undoubtedly derived from and closely related to the coccidia.

The various members of the class Sporozoa, as the name implies, produce at some stage of their development resistant spores within which occur one or more sporozoites, the actual infective forms. The latter, protected by the resistant capsule of the spore, are responsible for the carriage of an infection from one host to another. The term spore is, however, a very indefinite one, even apart from the fact that it is employed for resistant forms amongst bacteria and plants. If the term spore is regarded as being synonymous with the term sporocyst, as some writers maintain, then it would have to be admitted that some Sporozoa do not produce spores, though resistant encapsuled forms which are oöcysts occur. On this account it is better not to employ the term spore as indicating any particular stage of development, and it will be used merely as a name for any resistant stage of a parasite.

The life-cycles of many Sporozoa are characterized by an *alternation of generations*. A period of repeated asexual multiplication (schizogony) terminates in the production of gametes. The latter, by a process of conjugation (syngamy), give rise to zygotes, which multiply (sporogony) to form the infective stages (sporozoites), which are enclosed in resistant capsules. An *alternation of hosts* may also occur, in which case the asexual multiplicative phases may take place in one host and the sexual phases in another, while the infective forms, no longer exposed to the dangers of desiccation, may or may not be provided with a resistant

capsule, since they pass directly from one host to another without at any stage being outside the body of one or other host. The great majority of Sporozoa are intracellular parasites, at least during part of their growth period, and they obtain nourishment by absorption of the fluids of their hosts. The cells in which they live are, as a rule, irreparably damaged, so that they are to be regarded as definitely harmful organisms which are truly parasitic. In many instances the extent of tissue destruction, to which must be added the damage caused by soluble toxins, is so great that the health of the host is so seriously impaired that death may result. In other cases the damage inflicted is slight, and no noticeable change can be detected in the condition of the host. Frequently a balance is struck between the host and the parasite, and by continual reparation of the damage done the host may appear to be in perfect health.

The infection of a new host is brought about by the introduction of one or more motile vermiform sporozoites, which almost always find their way to some particular type of cell, into the cytoplasm of which they penetrate. There they become more or less spherical, and commence growing by absorption of fluid nutriment at the expense of the cell. During the early part of the growth period the nucleus of the parasite remains single, but either during growth (Coccidiomorpha) or after growth is complete (Gregarinina), nuclear multiplication takes place by repeated mitotic divisions, followed by division of the cytoplasm into a number of daughter individuals corresponding with the number of nuclei. These daughter individuals are either all gametes, which by conjugation (syngamy) produce zygotes which give rise to sporozoites again, or they are merozoites, which become adults without conjugation. In the latter case the production of merozoites is repeated a number of times till finally certain merozoites grow into gametocytes, which break up into gametes. These, as in the first instance, give rise after conjugation to sporozoites. It will be seen that two alternatives occur. The sporozoite either grows directly into a gametocyte which produces gametes **(Gregarinina)**, or it becomes a schizont which produces merozoites for several generations **(Coccidiomorpha)**. The final merozoites then become gametocytes, which give rise to gametes. In other words, there is either a sexual reproduction alone or an alternation of asexual and sexual reproduction.

In typical Sporozoa spore formation is associated with the process of conjugation or sporogony. The zygote resulting from the union of the male and female gametes becomes enclosed in a resistant cyst known as the oöcyst. Within this it may divide directly into a number of sporozoites, so that the mature *oöcyst* will contain a number of motile *sporozoites* and a residual body. On the other hand, the zygote may first divide into a number of separate bodies known as *sporoblasts*. These may become

encysted in secondary cysts called *sporocysts,* within each of which the
sporoblast gives rise to a number of sporozoites and a residual body.   In
such cases the mature oöcyst contains a number of sporocysts, each of
which has sporozoites within it.   When the zygote divides directly into
sporozoites, the oöcyst is known as *asporocystid,* while if sporocysts are
present, it is termed *sporocystid.*   If two, four, or more sporocysts are
present, it is called *disporocystid, tetrasporocystid,* or *polysporocystid.*   As
regards the sporocysts, they are known as *monozoic, dizoic, tetrazoic,* or
polyzoic, according to whether there are one, two, four, or more sporo-
zoites within each.   Thus *Eimeria schubergi,* the coccidium of the centipede,
which has oöcysts containing four sporocysts, each of which has two
sporozoites, is known as a dizoic tetrasporocystid coccidium.   The typical
gregarines produce asporocystid oöcysts, which contain eight sporozoites.
The oöcyst is typically seen amongst those Sporozoa which have only one
host, and in which infection is spread by escape of the oöcysts from the
body.   In such cases it is a highly resistant structure, which enables the
contents to withstand drying or other adverse influences.   Amongst those
Sporozoa which have two hosts, and in which external exposure of the
oöcyst does not occur, the oöcyst is not such a resistant structure, and is
modified in various ways.

SUBDIVISION OF THE SPOROZOA.

Doflein (1901) divided the Sporozoa into two orders, the Coccidio-
morpha and the Gregarinida (Lankester, 1866), but it seems better to
raise these to the rank of sub-classes.

1. *SUB-CLASS:* **Coccidiomorpha.** — The Sporozoa belonging to
this sub-class are nearly always intracellular during the whole of the
growing period. Asexual reproduction by repeated schizogony occurs, and
gametocytes are eventually produced.   The female- or macro-gametocyte
gives rise to one female- or macro-gamete, while the male- or micro-
gametocyte gives rise to a number of male- or micro-gametes, which are
very much smaller than the macrogametes.   Conjugation then takes
place between gametes, which are very unequal in size (anisogamy).

2. *SUB-CLASS:* **Gregarinina.**— The Sporozoa belonging to this
sub-class are typically intracellular only at the early part of the growth
period.   They then leave the cell and develop into more or less elongate
motile adults (gregarines).   Asexual reproduction does not occur except
in the small group of the Schizogregarinida.   The adult gregarines,
having developed directly from sporozoites, are therefore gametocytes.
These, however, do not show that differentiation into male and female
gametocytes which is a characteristic feature of the Coccidiomorpha.
The gametocytes associate in pairs, and the two produce an equal number

of gametes which are usually equal in size, though not always in character, since those arising from one gametocyte may be distinguished as female gametes, and those from the other as male gametes. Conjugation therefore takes place between similar or dissimilar gametes (isogamy or anisogamy).

1. *SUB-CLASS:* Coccidiomorpha Doflein, 1901.

The sub-class Coccidiomorpha includes two types of Sporozoa which differ in the method of conjugation. In one group (Coccidiida) the male and female gametocytes are of approximately equal size, and are not associated with one another. The male gametes are produced in comparatively large numbers, since they may have to wander a considerable distance before discovering a female gamete which can be fertilized. In the other group (Adeleida) the male and female gametocytes are closely associated in pairs (*syzygy*) during at least the latter part of their growth period. The male gametocyte is much smaller than the female, and, in consequence of its close association with the female, it produces only a small number of male gametes. The Coccidiomorpha may thus be divided into two orders, the COCCIDIIDA and the ADELEIDA.

Description of Types of the Coccidiida and Adeleida.

Before proceeding further, it will be advisable to consider the life-history of *Eimeria schubergi* and *Adelea ovata*, both of which are parasites of the intestine of the little centipede, *Lithobius forficatus*, and which may be taken as types of the two subdivisions of the Coccidiomorpha.

Eimeria schubergi (Schaudinn, 1900).—This parasite, which was discovered by Schaudinn, is of interest as being the first coccidium whose life-history was completely worked out (Fig. 337). Schaudinn's account is accurate on the whole, but, as pointed out by Schellack and Reichenow (1913), it is probable that his interpretation of the maturation process of the macrogametocyte was not correct, as also his explanation of the origin of the gamete nuclei in the microgametocyte from chromidia.

The parasite occurs fairly frequently in the intestine of the common garden centipede, together with other coccidia, and it may be studied in the intestinal contents and in smears or sections of the gut itself.

The centipede is infected in the first place by eating casually one or more oöcysts which have escaped in the fæces of an already infected individual. In its gut the sporozoites escape from the sporocysts, which split into two parts, and then, being free within the oöcyst, they make their way through the micropyle, an opening or pore at the end of the oöcyst wall (Fig. 337, 20). They move about by gliding, bending, and constric-

tion movements till they reach the surface of the gut epithelium. Each sporozoite, which measures 15 to 20 by 4 to 6 microns, then penetrates a cell, and becomes retracted to an ovoid mass of cytoplasm measuring about 16 microns in length and less than half this in diameter. There is a central nucleus consisting of a nuclear membrane on which chromatin granules are distributed, but, as in the nucleus of the sporozoite, there is no karyosome. The ovoid body grows rapidly, increasing in length and thickness, and at the same time a karyosome is developed within the nucleus, apparently from two substances—an achromatic material or plastin and chromatin. Growth is completed in about twenty-four hours, when the ovoid body becomes spherical and has a diameter of about 20 microns. The single nucleus then divides into two, and by repeated divisions from thirty to forty nuclei are formed. In nuclear division the nucleus elongates, while the karyosome does the same. The latter becomes dumbbell-shaped, with one rounded end at each pole of the nucleus. The chromatin granules of the nucleus collect in two groups, one at each end of the elongating karyosome. The connection between the halves of the karyosome breaks down, and nuclear division is completed by constriction and division of the nuclear membrane. The nuclei arrange themselves on the surface of the parasite, and finger-like elevations of cytoplasm are formed, into each of which a single nucleus passes. These elevations increase in length, gradually absorbing the cytoplasm from which they are growing, till finally there is left a residuum of cytoplasm (*residual body*), to which are attached in a radiating manner a number of uninucleate bodies (*merozoites*), measuring about 15 microns in length and 5 microns in breadth. The whole of this growth and multiplication has taken place within the epithelial cell of the centipede's gut (Fig. 337, 1-4). The cell is eventually reduced to a mere membrane, with its nucleus distorted and displaced to one side. As will be seen from this description, the schizont completes its growth before nuclear multiplication commences, and this feature has been supposed to be characteristic of the coccidia. Reichenow (1921*a*), however, believes that in *E. schubergi* nuclear multiplication commences before the schizont is fully grown, and that growth and nuclear multiplication take place simultaneously, as occurs in many other coccidia. The merozoites break loose from the residual cytoplasm to which they were attached, and lie free within the remains of the host cell. The latter ruptures, and the merozoites escape into the lumen of the gut. The merozoite differs from the sporozoite in being shorter and broader, in having a cytoplasm differentiated into an anterior vacuolated and a posterior denser area instead of being more uniformly vacuolated, and in having a karyosome in the nucleus. The merozoites are motile, like the sporozoites, and they penetrate fresh cells in a similar manner.

Here they again grow into adults, and reproduce as before. This process of multiplication is repeated a number of times till many parasites are produced. The growing form, whether derived from a sporozoite in the first place or from a merozoite, was termed a *schizont* by Schaudinn, and the process of multiplication terminating in the production of merozoites he called *schizogony*. The cytoplasmic mass to which the merozoites are attached is known as the *residual body*. The term "schizont" refers to that form which will ultimately reproduce by schizogony, so that the sporozoite or merozoite, when it has settled down in a host cell, is already a schizont, and one may legitimately speak of a young schizont, a half-grown schizont, or a schizont in which nuclear multiplication or merozoite formation or schizogony is taking place. Though the majority of fully-grown schizonts of *E. schubergi* are of the dimensions already given, a certain number do not attain this size. Very little growth may take place, and the number of nuclei produced may be as low as four, in which case only four merozoites are formed. Moreover, the merozoites, instead of being 15 microns in length, may be very much smaller (5 to 6 microns).

After asexual multiplication has been in progress for about five days, during which four or five generations of merozoites have been produced, another process of development is initiated. Some of the merozoites, instead of growing into schizonts, become differentiated into two distinct forms—male- or micro-gametocytes and female- or macro-gametocytes. During its growth the microgametocyte is very similar to a schizont, and when full grown is of approximately the same size as the mature schizont (Fig. 337, 5-8). Instead, however, of nuclear multiplication taking place by repeated divisions, another process is described by Schaudinn. The karyosome discharges its chromatin granules into the periphery of the nucleus, while the nuclear membrane gradually disappears. The chromatin granules, now free in the cytoplasm as chromidia, pass to the surface of the parasite, leaving the remains of the karyosome at the centre. The granules collect into strings, and finally form a series of tangled groups of approximately equal size. Each group becomes more and more compact, and finally assumes a comma shape. The microgametocyte now has the appearance of a spherical mass of cytoplasm, over the surface of which are arranged a number of comma-shaped nuclei which stain very deeply. Finally, each nucleus is separated with a small amount of cytoplasm, and the *microgamete* results. It measures 6 to 7 microns in length and barely 1 micron in breadth. It is rounded anteriorly and tapering posteriorly, and is provided with two flagella, an anterior one which trails behind in movement and a posterior one which appears to be a continuation of the posterior end of the microgamete, but which in all probability arises from the blunt end and passes backwards to the tapering posterior
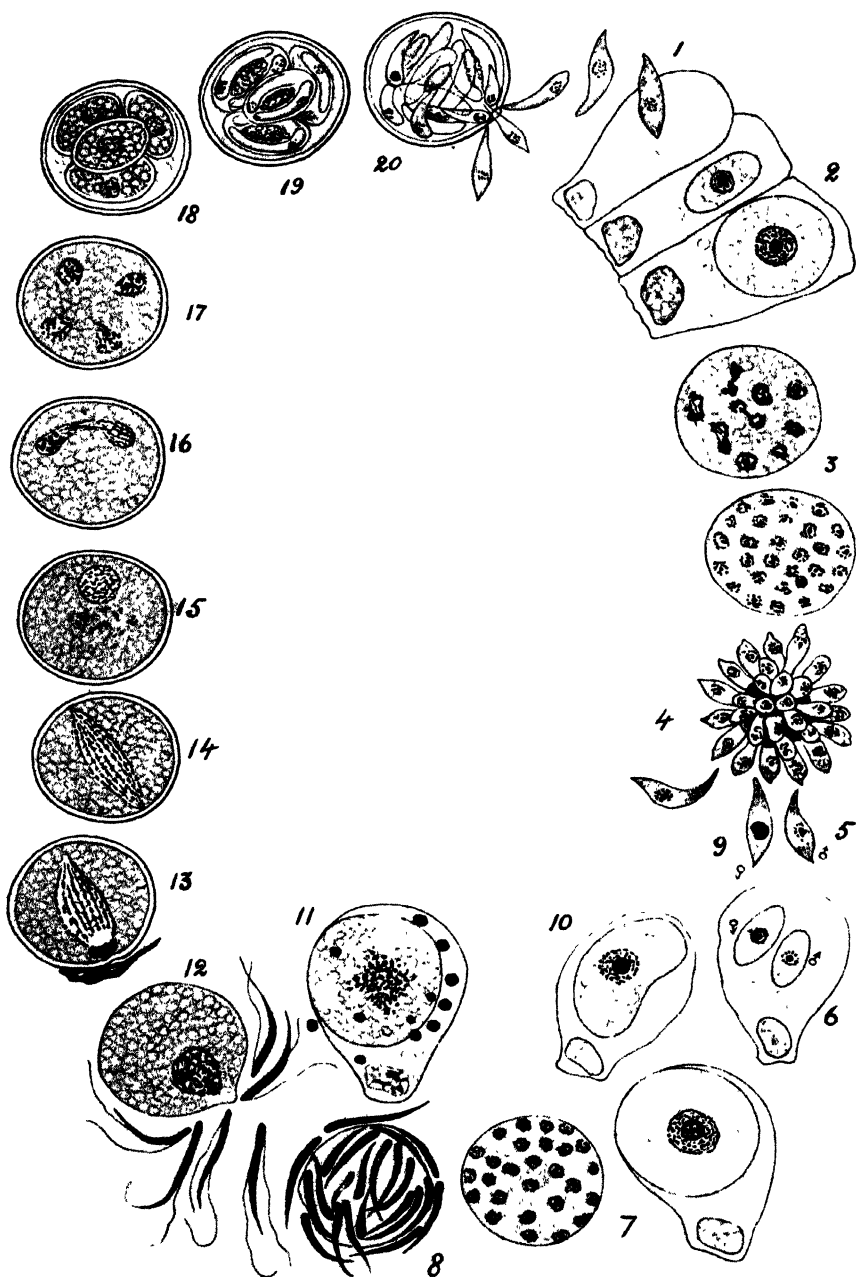
Fig. 337.—Life-Cycle of *Eimeria schubergi* in the Centipede, *Lithobius forficatus*. (After Schaudinn, 1900.) (× ca. 800.)

end over the surface of the body. The flagella are as long as or longer than the body. The microgamete corresponds with the spermatozoon of higher animals, and is destined to fertilize the female gamete when it escapes into the lumen of the gut after rupture of the host cell. The biflagellate nature of the microgametes of coccidia was first demonstrated by Wasielewski (1898) for the coccidium of the rabbit and a parasite of the centipede (probably *E. lacazei*). In the production of microgametes the bulk of the cytoplasm of the microgametocyte, together with the remains of the karyosome, is left over as a residual body, which degenerates. Considerable doubt, however, has arisen as to the accuracy of Schaudinn's account of the development of the microgametocyte. Schellack (1912, 1913) and Schellack and Reichenow (1913, 1915) have studied *E. schubergi* and other coccidia of the centipede, and find that the numerous nuclei which finally occur over the surface of the microgametocyte arise during its growth by repeated divisions of the original merozoite nucleus. No such process as the breaking up of the nucleus into chromidia and their condensation into a number of separate nuclei occurs. In the case of *Isospora felis* of the cat, the nuclei of the microgametes are formed by repeated mitotic divisions (Fig. 347, 12-15).

The young macrogametocyte is formed from a merozoite, as in the other stages described above. In this case, however, growth proceeds more slowly, and the cytoplasm becomes charged with a number of refractile globules, which may measure as much as 2 microns in diameter (Fig. 337, 9-11). The formation of this material in the cytoplasm differentiates the growing macrogametocyte from other stages. When fully grown, the macrogametocyte is an ovoid or bean-shaped body measuring about 30 microns in length and 10 microns in breadth. Its cytoplasm is filled with globules of the refractile material (food reserve), while the nucleus with its karyosome is at the centre. It then contracts gradually, becomes spherical, and escapes from the host cell. At a certain stage in this

1. Entry of sporozoite into epithelial cell of centipede's intestine.
2-4 Growth of schizont and production of merozoites by schizogony. The merozoites enter other epithelial cells and repeat the cycle 1-4.
5-8. Growth of microgametocyte and production of microgametes.
9-11. Growth of macrogametocyte. By the extrusion of the karyosome the macrogametocyte becomes a macrogamete.
12 Fertilization of the macrogamete.
13. The macrogamete nucleus is forming a spindle, at one end of which the microgamete nucleus lies. The oöcyst has formed.
14. The two nuclei have completely united owing to the distribution of the chromatin of both nuclei on the fertilization spindle.
15. The fertilization spindle has retracted to form the zygote nucleus.
16-17. Division of the zygote nucleus to form the four nuclei of the sporoblasts. It is probable that at the first of these divisions the number of chromosomes is halved.
18. The sporoblasts have been formed and sporocysts secreted around them.
19. Mature oöcyst with four sporocysts, each with two sporozoites and a residual body. At this stage the oöcysts escape from the intestine and are eaten by another centipede.
20. Sporozoites escaping from the sporocysts and oöcysts to infect the intestinal epithelial cell.

process the karyosome moves towards the nuclear membrane, passes through it into the cytoplasm, and breaks up into numerous granules, which are extruded from the surface of the parasite. This process Schaudinn claims to have observed several times in the living organism, and to have controlled by a study of stained preparations. The extrusion of the karyosome was regarded as a maturation process, and was supposed to correspond with the formation of polar bodies in the ova of higher animals. After extrusion of the karyosome, the microgamete has a marked affinity for the now mature macrogamete. Whatever may be the real explanation of the behaviour of the karyosome, the process does not correspond with the production of polar bodies, for it is almost certain that reduction in the number of chromosomes in the nucleus will be found to take place during the first division of the nucleus of the zygote, as occurs in other Coccidiomorpha (p. 109). By this process the macrogametocyte is converted into the *macrogamete*, and is ready for fertilization by a microgamete. The nucleus of the macrogamete after the extrusion of its karyosome appears to lose its nuclear membrane, so that its chromatin granules are spread through the central area of cytoplasm. It now becomes more compact and moves towards the surface, where a small elevation of the cytoplasm occurs. The nucleus itself is also drawn out slightly towards this point. A number of male gametes, which are attracted towards the female gamete only after the maturation process is complete, approach the cytoplasmic elevation, and one of these enters a slight depression at its apex (Fig. 337, 12). In so doing it is deprived of its flagella. The nucleus of the macrogamete now elongates, and a spindle is formed (*fertilization spindle*), at one end of which the nucleus of the microgamete, now become less compact and converted into a group of chromatin granules, takes up its position. The granules remain in this situation for some time, but finally pass into the spindle and mingle with the chromatin granules which were derived from the macrogamete nucleus. In this manner is brought about a union of the nuclei of the micro- and macrogametes. Syngamy is said to have taken place. Immediately after entry of the male gamete the cytoplasmic elevation is retracted, and a cyst wall of gradually increasing thickness, the oöcyst, is formed around what is now the zygote (Fig. 337, 13). The fertilization spindle remains as such for some time, but it finally retracts to form a spherical nucleus, which takes up a lateral position in the cytoplasm. It is at this stage that the oöcyst escapes to the exterior in the fæces of the centipede, the further development taking place in the ground.

The nucleus of the encysted zygote now becomes elongated and divides, and each daughter nucleus divides again (Fig. 337, 14-17). From researches of recent years upon the coccidia it would appear that, at the

first of these divisions, the number of chromosomes in the nucleus, which was doubled by the entry of the microgamete, is again reduced to half this number, and that the first division of the zygote nucleus (synkarion) is the real reducing division (p. 109). At division of the synkarion the chromosomes without dividing separate into two groups, one of which passes to each daughter nucleus. In the succeeding division, and, in fact, in all nuclear divisions of other stages of the cycle, the individual chromosomes divide, so that each daughter nucleus receives an equal number of divided chromosomes. This being the case, the maturation process, as described by Schaudinn, is not comparable with the formation of polar bodies, and probably has some other significance, as pointed out by Schellack and Reichenow (1913), who have noted that in the case of *Barrouxia schneideri*, *Adelea ovata*, *Adelina dimidiata*, *Eimeria lacazei*, and *E. schubergi*, all of them parasites of centipedes, there is no such process as maturation of the gametocyte by extrusion of the karyosome.

After nuclear division has been completed within the oöcyst, there are present four nuclei. The cytoplasm then segments into four portions (*sporoblasts*), while a small amount is left over as a residual body, in which are found certain granules, possibly of a chromatin nature, which were present in the cytoplasm. The sporoblasts are elongate ovoid bodies measuring about 12 by 7 microns. The cytoplasm contains the refractile globules present in the earlier stages. Each sporoblast now forms around itself a cyst (*sporocyst*), which consists of two elongated convex valves joined longitudinally (Fig. 337, 18). Within the sporocyst the sporoblast continues its development. The globules of refractile material run together to form two large globules, one at each end of the sporoblast. The nucleus divides by elongation and constriction, and this is followed by division of the cytoplasm into two *sporozoites* and a comparatively large residual body, which contains the unused portions of the food reserve material, which, after nuclear division, has collected at the centre of the sporoblast (Fig. 337, 19). The development of the oöcyst up to the formation of sporozoites occupies two to three days. It is now ready for ingestion by a new host.

The cycle of development involving the formation of the gametes, the fertilization, and the subsequent multiplication to form sporozoites, is known as *sporogony*, to distinguish it from the asexual cycle or *schizogony*, while the gametocytes are sometimes called male or female sporonts.

It will be seen that the sexually produced sporozoite always becomes a schizont, whereas the asexually produced merozoite may become one of three things—a schizont, a microgametocyte, or a macrogametocyte. From what is known of other coccidia, it seems very probable that all the merozoites which are derived from one parent by schizogony have a similar

fate. What kind of stimulus it is which determines the growth of a mero-zoite into schizont or gametocyte is not known, but the production of the gametocyte is essential for the maintenance of the species, as it is in association with sporogony that the resistant transmitting phases are produced. It is possible that all the merozoites derived from one schizont behave in their subsequent development in the same manner, and that the factor which determines whether they are to become schizonts again or gameto-cytes was already present before actual schizogony had taken place. In some coccidia, as will be seen below, the merozoites which are to become schizonts differ morphologically from those which are to become male or female gametocytes, and they are derived from schizonts which differ from one another also. That is to say, the merozoites which will become schizonts again are derived from schizonts, which differ morphologically from those schizonts which give rise to the merozoites which are destined to become gametocytes. In the case of *Cyclospora caryolitica*, Schaudinn describes this differentiation as carried further (Fig. 341). The sporo-zoites produce two kinds of schizont, each of which produces a particular kind of merozoite, which again become schizonts of the type from which they were derived. In this manner two distinct lines of repeated schizo-gony occur. Eventually the merozoites of one line become macrogameto-cytes, and those of the other microgametocytes. The sexual dimorphism in this case occurs from the beginning of the schizogony process. Reiche-now (1921a), however, doubts the accuracy of this observation.

Adelea ovata Ai. Schneider, 1875.—Like *E. schubergi*, which has just been considered, this coccidium is also a parasite of the centipede *Lithobius forficatus*. It was discovered and named by Aimé Schneider (1875) and was studied by Schaudinn and Siedlecki (1897), Siedlecki (1899a), Dobell (1907), Jollos (1909), Debaisieux (1912), Greiner (1918), and Schellack and Reichenow (1910, 1915). The last named observers have given the most complete account of its development, the main outlines of which resemble that of *E. schubergi* (Fig. 338). The entry of a sporozoite into an intestinal cell initiates a period of reproduction by schizogony, which is followed by the production of microgametes and macrogametes. Fertiliza-

1-5 Schizogony in gut epithelium of the centipede.   6. Schizogony producing larger merozoites.
7. Young male gametocyte.   8 Young female gametocyte.
9. Association of male with very much enlarged female gametocyte.
10. Nucleus of male gametocyte dividing to produce four microgamete nuclei. Female game-tocyte mature.
11. Male gametocyte has produced four male gametes, one of which has entered the female gamete and broken up into granules. The oöcyst has formed.
12. Later stage: one male gamete has entered the female gamete, while three remain outside the oöcyst. Fertilization spindle is commencing to form.
13. Stage showing the fertilization spindle.
14. Fully-formed zygote in oöcyst.   15. Multiplication of nuclei of zygote (sporont).
16. Mature oöcyst containing numerous sporocysts, each of which has two sporozoites and a residual body.

FIG. 338. LIFE-CYCLE OF *Adelea ovata*. (COMPILED WITH SLIGHT MODIFICATIONS FROM FIGURES OF SCHELLACK AND REICHENOW, 1915). (× 1,200.)

tion takes place, and there is produced an oöcyst containing sporocysts and sporozoites. In details the cycle differs, however, from that of *E. schubergi* in some important aspects, but chiefly in the close association (syzygy) of the micro- and macro-gametocytes, and the production by the former of only a small number of microgametes (Fig. 338, 9-11).

Infection of the centipede is brought about by the ingestion of ripe oöcysts, and the liberation of sporozoites in its intestine by a separation of the two valves of which the sporocyst is composed. These enter the intestinal cells, retract to a spherical form, and commence growing at the expense of the cell cytoplasm. As growth proceeds, the nucleus multiplies by repeated divisions till as many as twenty to fifty are present. Segmentation of the schizont, which measures from 40 to 70 microns in its longest diameter, into a corresponding number of elongate vermiform merozoites then takes place (Fig. 338, 1-5). A residual body may or may not be present. When present, it varies considerably in size. Sometimes several bodies occur. By rupture of the cell the merozoites are set free in the lumen of the gut, whence they invade other cells, and the process of schizogony is repeated. After several generations merozoites become differentiated into micro- and macro-gametocytes (Fig. 338, 7-9). The macrogametocyte increases markedly in size, while the karyosome within its nucleus also augments its volume. The microgametocyte, on the other hand, only increases slightly in size, while its karyosome remains small. Early on in this process of growth the microgametocyte becomes applied to the surface of a macrogametocyte, but no actual union takes place (Fig. 338, 9). The two gametocytes are then said to be in syzygy. When fully grown the macrogametocyte has an ovoid form, while the large karyosome within the nucleus becomes smaller, and finally disappears. It is not thrown out, as described in *E. schubergi*. The nucleus then moves to one end of what is now the macrogamete. Meanwhile, the microgametocyte has become spherical, and by two divisions the single nucleus has become four. The four nuclei give rise to four comma-shaped microgametes, each of which, from what is known of other coccidia, is probably provided with two flagella, though they have not yet been demonstrated in *Adelea ovata* (Fig. 338, 10). One of the microgametes penetrates the macrogamete at the end near which its nucleus is now lying and enters the nucleus. The fertilized macrogamete, which is now the zygote, secretes around itself the oöcyst, while its nucleus (synkarion), containing the elements from the micro- and macro-gamete nuclei, moves towards the centre (Fig. 338, 11). The nucleus then becomes elongated to form a spindle (fertilization spindle), while the chromatin becomes arranged in the form of thread-like chromosomes lying parallel to one another (Fig. 338, 12-13). When this has occurred the spindle retracts, and at the

same time the chromosomes become shorter and plumper, till finally a spherical nucleus with short chromosomes results. The nucleus which has become spherical proceeds to division, and Reichenow (1921) states that at the first division each daughter nucleus receives half the chromosomes, which do not themselves divide. In this manner the number of chromosomes in the nucleus is halved. This is the real reduction division. Schellack and Reichenow (1915) point out that the changes undergone by the karyosome of the macrogametocyte nucleus have no connection with the process of maturation. The two daughter nuclei thus formed divide again, but in this case the chromosomes themselves divide, so that the number is maintained. Repeated divisions of this kind take place till sixteen to thirty-two nuclei are present (Fig. 338, 15). The cytoplasm then buds off from its surface a corresponding number of spherical sporoblasts, which secrete around themselves the bivalved sporocysts, which are discoidal and flattened like two applied watch-glasses. The nucleus of each sporoblast divides, and there are formed within each sporocyst two sporozoites and a residual body (Fig. 338, 16). Contrary to what occurs in *E. schubergi*, the oöcysts are completely developed when passed in the fæces of the centipede. The nucleus of each sausage-shaped sporozoite is terminal, while its cytoplasm contains a large hyaline body.

Subdivision of the Coccidiomorpha.

It will be noted that the cycle of development described above for *Adelea ovata* differs from that of *Eimeria schubergi* in the fact that the microgametocyte is smaller than the macrogametocyte, and that the two are in close association (syzygy). Furthermore, the number of microgametes produced is only four instead of a large number. The close association of the gametocytes renders it easier for the microgamete to discover the macrogamete than if the gametocytes were far apart, as in *E. schubergi*. A large number of coccidia, the hæmosporidia, and a few of the hæmogregarines, behave in this respect like *E. schubergi*, while others, including most of the hæmogregarines, behave like *A. ovata*. On this account the sub-class Coccidiomorpha is divisible into two orders— the COCCIDIIDA, in which fertilization is of the *E. schubergi* type; and the ADELEIDA, in which it follows *A. ovata*. These two orders are again divisible into sub-orders. The Coccidiida includes the two sub-orders **Eimeriidea** and **Hæmosporidiidea**. A member of the Eimeriidea is characterized by the fact that the microgametocyte produces flagellated microgametes by a slow process of nuclear multiplication, and that the zygote resulting from conjugation is already of its maximum size, is not motile, and at once becomes enclosed in a tough oöcyst, which retains

its shape and size, and is destined to protect the sporozoites from damage due to desiccation and exposure after escape from the body of the host. A member of the **Hæmosporidiidea** is characterized by the fact that the microgametocyte produces non-flagellated microgametes by a suddenly occurring violent process of extrusion (flagellated bodies), and that the zygote becomes a motile vermicule (oökinete). The oökinete eventually forms an oöcyst, which is not a resistant structure, for it increases in size after it is first formed, and is not destined to protect the sporozoites from exposure, for they escape from the oöcyst very soon after they are developed. This lack of a tough oöcyst is associated with a development in two hosts in such a manner that at no stage is the parasite outside one or other host.

The order Adeleida includes the sub-orders **Adeleidea**, in which a tough resistant oöcyst, which maintains its shape and size, is developed for protection of the sporozoites during external exposure; and the sub-order **Hæmogregarinidea**, in which no such resistant oöcyst is produced on account of the existence of a second invertebrate host in which the sporogony cycle takes place. In the Adeleidea, again, the macrogametocyte is a stationary body to which the microgametocyte early applies itself, while in the Hæmogregarinidea the gametocytes are motile vermicules, which wander about in the stomach of the invertebrate till they associate in pairs, which still travel as double vermicules till they settle down for further development. It will thus be seen that in the sub-orders Eimeriidea and Adeleidea tough resistant oöcysts are produced, as there is only a single host, and the gametocytes and zygotes are stationary bodies; in the sub-orders Hæmosporidiidea and Hæmogregarinidea, in which there are two hosts, the tough oöcyst as a permanent protective structure is not developed, while the gametocytes or zygotes are definitely motile.

There is a group of blood parasites, commonly known as the piroplasmata, which occupy the red blood-corpuscles of mammals, are transmitted by ticks, and resemble in many respects the hæmosporidia. In no case, however, is the life-cycle completely known, so that, apart from the observations of Christophers on *Babesia canis*, which have not been confirmed, there are no data as to the method of fertilization nor of the development in the invertebrate hosts. It is possible that the association of the gametocytes of *B. canis* in the tick, which he has described, indicates a process of syngamy similar to that characteristic of the Adeleidea, but the details have not been worked out. On this account it is impossible to classify them with any certainty. They will be placed in a provisional sub-order of the Coccidiomorpha, the **Piroplasmidea**, next to the Hæmosporidiidea.

The investigations of the past few years have shown quite clearly that the blood-inhabiting Sporozoa have been derived from the coccidia, which are typically intestinal parasites. Mesnil (1899) was the first to indicate clearly that the " hæmocytozoa," as the parasites of the blood-cells have been called, should be grouped with the coccidia. Schaudinn (1899) also drew attention to the close similarity of the life-cycles of the malarial parasites and coccidia, while Reichenow (1912) definitely regarded them as " hæmococcidia." They are undoubtedly coccidia which have become modified for a life in the circulating cells of the blood, and in association with this change the extracorporeal phase seen in the coccidia, which have only one host, is passed in the body of an invertebrate, which eventually transfers the parasite back again to the vertebrate. Of these blood coccidia there are two types. Firstly, the Hæmosporidiidea, which have evolved from those coccidia which have a fertilization of the *Eimeria* type, as described above; and, secondly, the Hæmogregarinidea, which have originated from coccidia, in which fertilization is of the *Adelea* type.

Schaudinn (1904) suggested that a definite relationship existed between the hæmosporidia and the trypanosomes, and he even went so far as to describe definite trypanosome phases in the development of *Hæmoproteus* and other Hæmosporidiidea. Though Schaudinn's observations were undoubtedly incorrect, his views were accepted in certain quarters. Thus we find Hartmann (1907) and Hartmann and Jollos (1910) separating the hæmosporidia from the Sporozoa, and placing them in a separate order (Binucleata) of the Mastigophora. Apart from the observations which Schaudinn claimed to have made, the view is based on the assumption that the trypanosomes possess two nuclei, and that many hæmosporidia show the same two nuclei. There seems to be no ground to justify the conclusion that the nuclear arrangement in the Hæmosporidiidea is homologous with that of a trypanosome, and in view of the overwhelming evidence in favour of regarding the hæmosporidia as having originated from coccidia, Hartmann's view cannot be accepted. Alexeieff (1910a) pointed out that Hartmann's group, Binucleata, was a purely artificial one.

Taking into consideration the facts explained above, the sub-class, Coccidiomorpha, may be subdivided as follows:

1. *Order:* COCCIDIIDA Labbé, 1899.—During the whole of their growth the male and female gametocytes are apart, and develop independently of one another. The male gametocyte produces a relatively large number (six or more) of male gametes.

(1) *Sub-Order:* **Eimeriidea.**—The male gametes are formed on the surface of the male gametocyte, which has become multinucleate owing to repeated divisions of the nuclei during its growth. The motionless

zygote secretes a resistant oöcyst, which does not increase in size. The asexual and the sexual cycles occur in one host.

(2) *Sub-Order:* **Hæmosporidiidea.**—The fully-grown male gametocyte, which has a single nucleus, produces male gametes by a violent process of flagellation. The zygote becomes a motile oökinete, which finally secretes an oöcyst which increases in size. There is an alternation of hosts, the asexual cycle occurring in a vertebrate and the sexual cycle in an invertebrate.

(3) *Sub-Order:* **Piroplasmidea.**—The exact method of syngamy is not known. It appears that the zygote becomes a motile oökinete, and that the asexual cycle occurs in a vertebrate host and the sexual cycle in an invertebrate.

2. *Order:* ADELEIDA.—During their growth male and female gameto-cytes become closely associated and develop in contact with one another. The male gametes, which are few in number (two or four), are formed on the surface of the male gametocyte, which has become multinucleate owing to divisions of the nuclei after association has taken place.

(1) *Sub-Order:* **Adeleidea** Léger, 1911. — The motionless zygote becomes enclosed in a resistant oöcyst, which does not increase in size Both the asexual and the sexual cycles occur in one host.

(2) *Sub-Order:* **Hæmogregarinidea.**—The zygote is a motile oökinete which forms an oöcyst which increases in size. There is an alternation of hosts, the asexual cycle occurring in a vertebrate host and the sexual cycle in an invertebrate.

1. *Order:* COCCIDIIDA.

(1) *Sub-Order:* **Eimeriidea.**

Subdivision into Families.

The sub-order Eimeriidea contains a number of families, which are distinguished from one another by variations in the development of the oöcyst and in details of the schizogony cycle. The following families are included:

1. *Family:* SELENOCOCCIDIIDÆ Poche, 1913.—The sporozoite does not enter a cell, but grows in the lumen of the intestine into an elongate actively motile vermiform organism. The nucleus multiplies to form eight, and then the schizont enters a gut cell to become spherical and break up into eight merozoites, which are discharged into the lumen of the gut. The growing phases of the schizont are thus free extracellular organisms. Similarly the micro- and macro-gametocytes are formed from merozoites extracellularly, and have vermiform bodies. They enter cells

and become rounded as micro- and macro-gametocytes. The former produces a large number of microgametes, one of which fertilizes a macrogamete, and an oöcyst, the development of which has not been followed, is then formed.

2. *Family:* CRYPTOSPORIDIIDÆ Poche, 1913. — These are minute coccidia which inhabit the intestine of mice. The whole cycle is passed on the surface of the gut cells. There is a cycle of schizogony, followed by production of micro- and macro-gametocytes and formation of oöcysts. The latter, however, are asporocystid, and produce sporozoites without sporocysts. The schizonts, as well as the gametocytes, appear to grow within delicate cysts, to one end of which the cytoplasm is attached. When merozoites or microgametes are produced within these cysts, they arise on the surface of the cytoplasm opposite the point of attachment.

3. *Family:* EIMERIIDÆ Poche, 1913.—The members of this family include the typical coccidia, like *Eimeria schubergi.* Schizogony and sporogony are very uniform in character, except that variations occur in the development of the oöcyst. In some cases sporocysts are not produced within the oöcyst (asporocystid), in some two or four are produced (disporocystid, tetrasporocystid), while in other cases many occur (polysporocystid). When sporocysts are present, the number of sporozoites within each is variable. There may be only one (monozoic), or two (dizoic), four (tetrazoic), or many (polyzoic). The family is divisible into a number of sub-families as follows:

(1) *Sub-Family:* CYCLOSPORINÆ.—The oöcyst, which is dizoic disporocystid, produces two sporoblasts and two sporocysts, within each of which are two sporozoites. The schizogony cycle commencing from the sporozoite is, according to Schaudinn, differentiated on two lines, there being two kinds of schizont, each producing a particular kind of merozoite. Eventually the merozoites of one kind give rise to microgametocytes and those of the other to macrogametocytes.

(2) *Sub-Family:* ISOSPORINÆ.—The oöcyst, which is tetrazoic disporocystid, produces two sporoblasts and two sporocysts, each of which develops four sporozoites. In other respects the cycle is typical.

(3) *Sub-Family:* EIMERIINÆ.—The oöcyst, which is dizoic tetrasporocystid, produces four sporoblasts and four sporocysts, each of which develops two sporozoites. The rest of the cycle is typical.

(4) *Sub-Family:* BARROUXIINÆ.—The oöcyst, which is monozoic polysporocystid, produces many sporoblasts and many sporocysts, in each of which only one sporozoite is formed. The other stages are typical.

(5) *Sub-Family:* CARYOSPORINÆ.—The oöcyst, which is polyzoic monosporocystid, produces only one sporoblast and one sporocyst, in which eight sporozoites are developed. The cycle is typical in other respects.

(6) *Sub-Family:* PFEIFFERELLINÆ.—The oöcyst, which is asporocystid, produces eight sporozoites without any sporocyst. The schizogony results in the production of a large number of merozoites, while at the time of fertilization the macrogamete extrudes a long cytoplasmic process for reception of the microgametes.

4. *Family:* CARYOTROPHIDÆ Lühe, 1906. — The oöcyst, which is polyzoic polysporocystid, produces many sporoblasts and many sporocysts, each of which contains many sporozoites. The asexual cycle is complicated in that the schizont divides into a number of intermediate bodies (*schizontoblasts, agametoblasts, cytomeres*), each of which subsequently produces merozoites. Similarly the microgametocyte first segments into a number of *microgametoblasts*, which then produce microgametes. The macrogametocyte, as usual, becomes a single macrogamete.

5. *Family:* AGGREGATIDÆ Labbé, 1899.—The oöcyst, which is polysporocystid, produces many sporoblasts and many sporocysts, each of which contains a small number of sporozoites. The oöcysts develop to maturity in the body of a cuttlefish which is eaten by a crab. The sporozoites which escape become schizonts in the gut wall of the crab, where schizogony occurs. Eventually the crab is eaten by a cuttlefish, when certain merozoites in the gut of the crab invade the gut wall of the cuttlefish and develop into micro- and macro-gametocytes. The oöcyst is formed in the usual manner in the gut wall of the cuttlefish. There is thus an alternation of hosts, in one of which schizogony occurs, while sporogony takes place in the other.

6. *Family:* LANKESTERELLIDÆ Reichenow, 1921.—The oöcyst is asporocystid, and produces within it a number of sporozoites without sporocysts. The schizogony cycle is of the usual type, and eventually there are produced micro- and macro-gametocytes. After fertilization the oöcyst is formed, and the sporozoites developed. The latter are liberated from the oöcyst, which does not leave the body of the vertebrate. They make their way to the circulating cells of the blood—usually the red blood-corpuscles—within which they appear as hæmogregarines. Without further development, beyond a slight increase in size, they are taken up by an invertebrate and transferred to another vertebrate, where the schizogony cycle commences again. These forms can be grouped in two sub-families—Schellackinæ and Lankesterellinæ.

(1) *Sub-Family:* SCHELLACKINÆ.—The whole development up to the ripening of the oöcyst takes place in the intestinal epithelium, with the exception that the macrogametocyte and oöcyst are formed in the subepithelial connective tissue. By rupture of the oöcyst the sporozoites are liberated in the subepithelial connective tissue and gain entrance to the

blood-vessels, where they enter the red blood-corpuscles. The invertebrate vector is a mite.

(2) *Sub-Family:* LANKESTERELLINÆ. — The whole development takes place in the endothelial cells lining the blood-vessels. By rupture of the oöcyst the sporozoites are set free in the blood-stream, when they enter red blood-corpuscles. The invertebrate vector is a leech.

Systematic Description of Families of the Eimeriidea.

These various families may now be considered in greater detail. The individual parasites reveal a marked uniformity in the asexual cycles, but variations occur in the development of the zygote (sporogony), in the character of the oöcysts, in the presence or absence of sporocysts, in the number of these when present, and the number of sporozoites each contains.

They are mostly parasites of the lining cells of the intestine of their hosts. Sometimes, however, they occur in other organs. Very rarely they are extracellular parasites. In the case of ordinary examinations of the fresh intestinal contents or voided fæces, it is the oöcyst which is usually recognized, while the remainder of the cycle can only be studied in preparations of the tissue in which it occurs. In the case of warm-blooded hosts such as man, the complete development of an oöcyst does not, as a rule, take place till after it has escaped from the body, possibly as a result of the stimulus of change in temperature. This being the case, it may be impossible to identify an oöcyst as such till the material in which it occurs has been kept at ordinary atmospheric temperature for a few days, when the formation of sporocysts and sporozoites will have taken place. On this account the oöcysts of coccidia have frequently been mistaken for the eggs of worms, which they resemble in the thickness of the capsule and the granular nature of the enclosed zygote. The oöcysts of coccidia, however, are generally considerably smaller than the eggs with which they have been confused. Sometimes the oöcysts from one animal will appear in the fæces of another. Thus the fæces of human beings will sometimes contain oöcysts from fish. In such cases they are present for a short time only, and must not be regarded as parasites of man (see p. 851).

The intestinal coccidia often produce extensive destruction of the epithelium and acute diarrhœa, or even dysenteric conditions, which may bring about the death of the host. The host may survive the acute phase of an infection and pass into a chronic one with a milder course. Many animals in apparently perfect health are found to harbour coccidia, and it is probable that, having survived an acute infection, they have adapted themselves to the parasite, and are able to repair the damage done to the cells as quickly as it takes place.
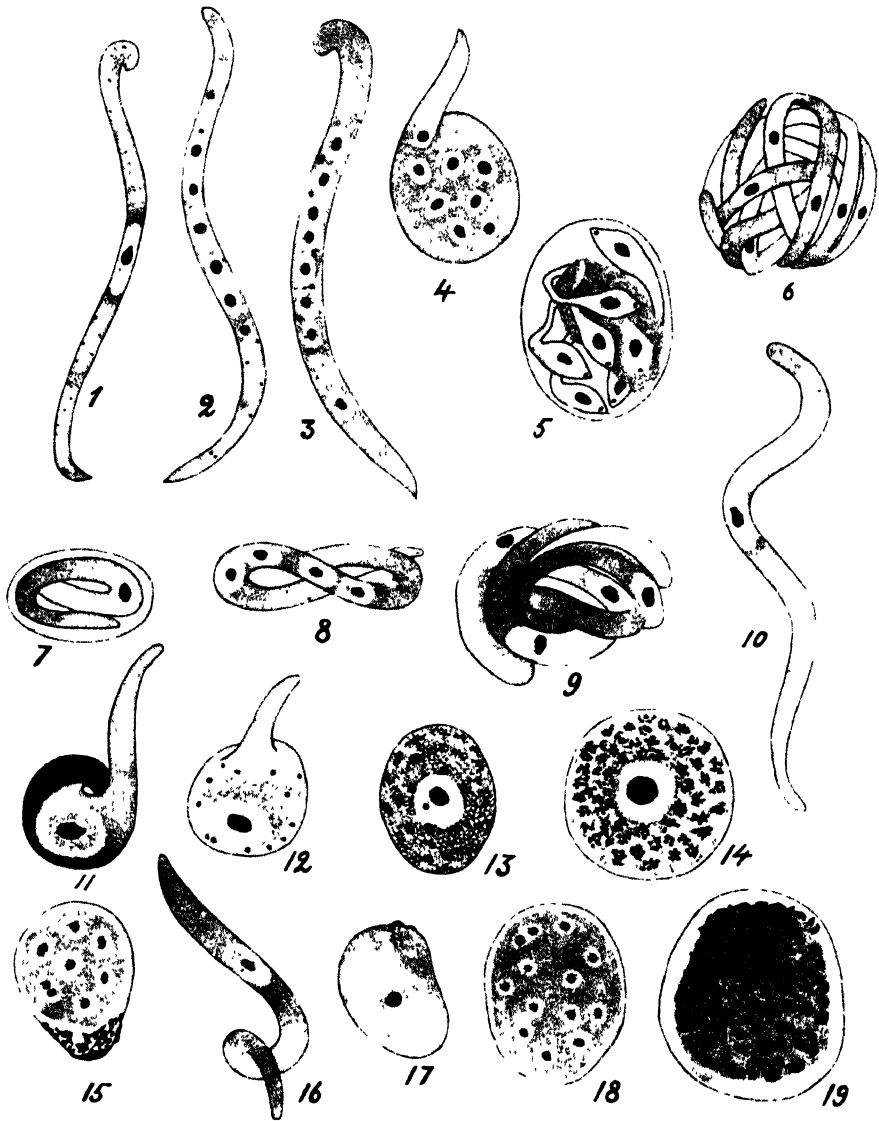
FIG 339.—*Selenococcidium intermedium* (× 850). (AFTER LÉGER AND DUBOSCQ, 1910.)

1. Vermicular schizont free in gut.
2-3. Vermicular schizonts showing nuclear multiplication.
4. Retraction of schizont to spherical form—intracellular.
5-6. Schizogony to form merozoites.
7-9. Schizogony to four merozoites, which become macrogametocytes.
10-12. Retraction of vermicule to form macrogametocyte.
13. Macrogametocyte.   14. Oöcyst.
15 16. Schizogony to form eight merozoites, which become microgametocytes.
16 19. Vermicule which becomes microgametocyte, producing microgametes.

1. *Family:* SELENOCOCCIDIIDÆ Poche, 1913.

This family includes the single genus *Selenococcidium* Léger and Duboscq, 1909, of which there is a single species.

Selenococcidium intermedium Léger and Duboscq, 1909.—This coccidium is a parasite of the intestine of the lobster. Its life-cycle has been described by Léger and Duboscq (1910). It is remarkable in that the growing forms, both schizonts and gametocytes, are elongate vermicules which live free in the intestine (Fig. 339). In the case of the schizont, nuclear multiplication takes place till eight nuclei are present. The vermicule (60 to 100 microns in length) then retracts to a globular form, and while doing so enters the gut cells, where it divides into a number of vermicular merozoites. These become free in the gut again, and grow into schizonts as before (Fig. 339, 1-6). After schizogony has been repeated a number of times two different types of schizont are produced. One is a small one, which produces eight small vermicules. After a free existence they enter cells, again become rounded, and develop into typical microgametocytes, which produce a large number of microgametes in the typical coccidian manner (Fig. 339, 15-19). The other is a larger schizont, which produces only four vermicular merozoites. These, after a free existence, enter cells and become macrogametes (Fig. 339, 7-14). After fertilization has taken place, there is formed a spherical oöcyst which escapes from the intestine. It is not known, however, how the oöcyst develops its sporozoites. *S. intermedium* is of considerable interest, as its free motile vermicule stage serves as a connecting link between the coccidia and the gregarines.

2. *Family:* CRYPTOSPORIDIIDÆ Poche, 1913.

In this family there is only one genus, which was founded by Tyzzer (1907). There are two species, *Cryptosporidium muris* Tyzzer, 1907, and *C. parvum* Tyzzer, 1912. The former is a parasite of the peptic glands of the mouse, and the latter of the small intestine of the same animal (Fig. 340).

Cryptosporidium muris Tyzzer, 1907.—This is a common parasite of white mice, and can often be seen in sections of the stomach in enormous numbers in the lumen of the glands. It is peculiar in not being intracellular at any stage of its development, the whole of which takes place in the mucoid material on the surface of the cells, the parasite appearing to be attached to the actual surface at one point. The organism is a very small one, and was first studied in detail by Tyzzer (1910) in sections of the stomach wall and in smears from its surface. The young schizont attached to the surface of a gland cell consists of a minute mass of cytoplasm with a single nucleus. It often appears as if it were surrounded by a cyst wall

or membrane, to one side of which the cytoplasm is attached. As growth takes place the nucleus multiplies. Finally, it reaches a maximum size of 7 by 6 microns and contains eight nuclei, which are arranged in the cytoplasm on the side opposite the point of attachment. Division into eight merozoites takes place. That schizogony is repeated many times seems to be indicated by the very large infections frequently encountered. As usual in the coccidia, microgametocytes and macrogametocytes are eventually formed. The former, when mature, measures 5 by 4 microns,
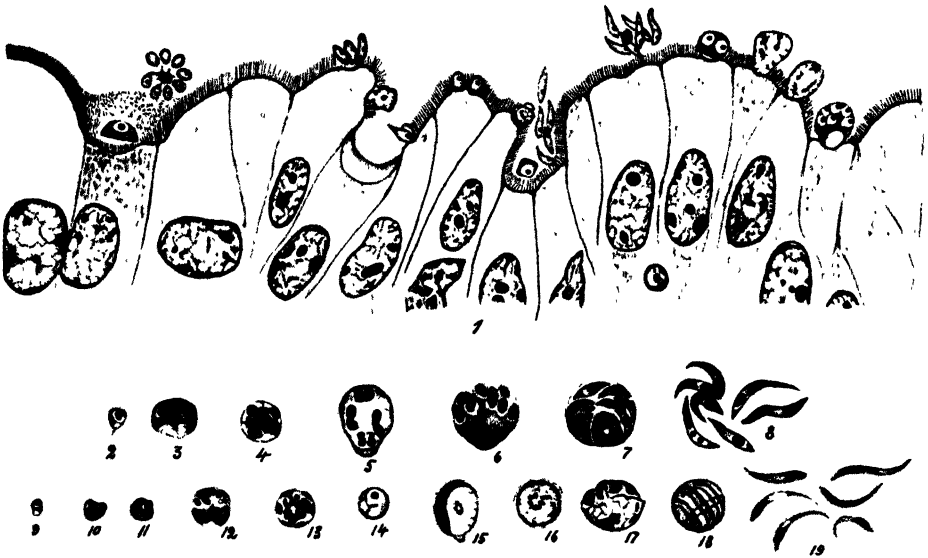
FIG. 340 —*Cryptosporidium parvum* FROM THE SMALL INTESTINE OF MICE (× *ca.* 2,000). (AFTER TYZZER, 1912.)

1. Diagram drawn up from Tyzzer's figures of section of intestine, showing various stages of the parasite on the surface of the epithelium.   2-8. Schizogony cycle.
9-13. Development of microgametocyte and formation of microgametes.
14-15. Development of macrogametocyte.
16-18. Development of oöcyst and formation of sporozoites.
19. Sporozoites escaped from oöcyst.

and has sixteen minute comma-shaped nuclei, from which sixteen microgametes are formed. The macrogamete measures about 7 by 5 microns, and consists of cytoplasm containing globules of hyaline material, as in coccidian macrogametes generally. After fertilization, which takes place without any association of the micro- and macro-gametocytes, a thick oöcyst is secreted, and within it the zygote divides after nuclear multiplication into four masses of cytoplasm, each with a single nucleus and a comparatively large residual body. Each of the four masses becomes elongated to form a slender sporozoite, which measures about 12 to 14 microns

in length. The sporozoites are slightly longer and narrower than the merozoites. The mature oöcyst has the same dimensions as the macro-gamete. Many of the oöcysts liberate their sporozoites while still in the stomach of the host in which they were developed, so that auto-infection, both with merozoites and sporozoites, probably takes place. On the other hand, oöcysts pass into the intestine and, escaping with the fæces, spread infection to other mice.

Cryptosporidium parvum Tyzzer, 1912.—Tyzzer (1912) has also described another and still smaller species (*C. parvum*) which is found in the glands of the small intestine of mice (Fig. 340). By feeding experiments Tyzzer has established the individuality of the two species. *C. parvum*, and possibly also stages of *C. muris*, were seen by the writer (1907) in mice, but he confused the forms he saw with *Eimeria falciformis*, with which the animals were also infected. In *C. parvum* the schizont attains a maximum diameter of 5 microns and produces eight merozoites, each of which measures from 2·5 to 5 microns in length by 0·5 to 0·7 microns in breadth, and has a nucleus situated near the anterior extremity. Many have a deeply staining granule in addition to the nucleus, and this enters into the attachment organ by which the merozoite fixes itself to the surface of the cell before commencing its growth into a schizont again. The micro-gametocyte, when fully grown, is not over 3 microns in diameter, and gives rise to sixteen microgametes. The macrogamete after fertilization produces an oöcyst with its longest diameter of not more than 4·5 microns. Within it are produced four sporozoites.

Both *C. muris* and *C. parvum* agree with one another in having an extracellular habitat. They both reproduce by schizogony, and eventually give rise to oöcysts in which the sporozoites are free, there being no secondary sporocyst formation.

Triffitt (1925a) has given the name *C. crotali* to a parasite of the snake, *Crotalus confluentus*. Ovoid cysts measuring 10·8 to 12·5 by 10 to 11 microns, and containing four sporozoites and residual material, were discovered in the fæces. No other stages of development were seen. It appears possible that the cysts were the sporocysts of a species of *Isospora*, but until further stages have been found it is evidently impossible to be certain of the actual genus to which the parasite belongs.

3. *Family:* EIMERIIDÆ Poche, 1913.

The various members of this family agree with one another in that the whole of the growth period is passed within the cytoplasm of a host cell. Schizogony with the production of merozoites takes place repeatedly. Finally, merozoites become differentiated into macro- and micro-gameto-cytes which, when fully grown, are almost equal in size. Large numbers

of microgametes are produced which fertilize the macrogamete. Fertilization usually takes place through a pore, the micropyle, which occurs at one end of the oöcyst. After fertilization, the micropyle is closed and the oöcyst completes its development in various ways.

<div align="center">(1) <i>Sub-Family</i> : CYCLOSPORINÆ.</div>

This sub-family includes the single genus *Cyclospora*, which was founded by Aimé Schneider (1881) for a parasite, which he named *C. glomerica*, of the myriapod, *Glomeris*. The best-known form, however, is *C. caryolytica* Schaudinn, 1902.

Cyclospora caryolytica Schaudinn, 1902.— This parasite was first noted by Eimer (1870) in the intestine of the mole. It was again seen by Grassi (1881a), while Schaudinn (1902) studied its life-history and gave it its name (Fig. 341). It develops in the intestinal epithelium, and produces a severe enteritis. The infection is initiated by the mole ingesting a mature oöcyst, which contains two sporocysts, each with two sporozoites. The sporozoites escape and invade the gut epithelium, where they penetrate to the interior of the nuclei and commence growth. In this process the nucleus is broken up and the cell reduced to a mere membrane enclosing the parasite. As growth takes place it is seen that two types of schizont are being produced, one which is large and composed of hyaline cytoplasm, and the other small and of denser cytoplasm (Fig. 341, 1-3, 6-8). The large schizont, when it breaks up after nuclear multiplication, produces clear stumpy merozoites, while the other gives rise to longer and more opaque merozoites. The former again grow into large schizonts after they have entered fresh nuclei, and again the same type of merozoite is produced, while the larger and denser merozoites again produce the smaller schizonts, which break up into the same type of merozoite again. There are thus two distinct lines along which schizogony occurs. Eventually merozoites of the short hyaline type become microgametocytes (Fig. 341, 4-5), while the long dense ones become macrogametocytes (Fig. 341, 9-10). The microgametocyte produces a large number of flagellate microgametes, while the macrogametocyte undergoes a maturation process. The latter differs from that described for *Eimeria schubergi*. The nucleus divides, and one of the resulting nuclei begins to degenerate. The other nucleus again divides, and again one of the nuclei disintegrates. The remaining nucleus is that of the mature macrogamete. A number of microgametes

1-3. Schizogony cycle, which ultimately leads to the formation of the microgametocytes.
4-5. Development of microgametocyte and formation of microgametes.
6-8. Schizogony cycle, which ultimately leads to the formation of the macrogametocyte
9-10. Development of macrogametocyte.   11. Fertilization.
12-14. Development of oöcyst with production of two sporocysts, each containing two sporozoites.
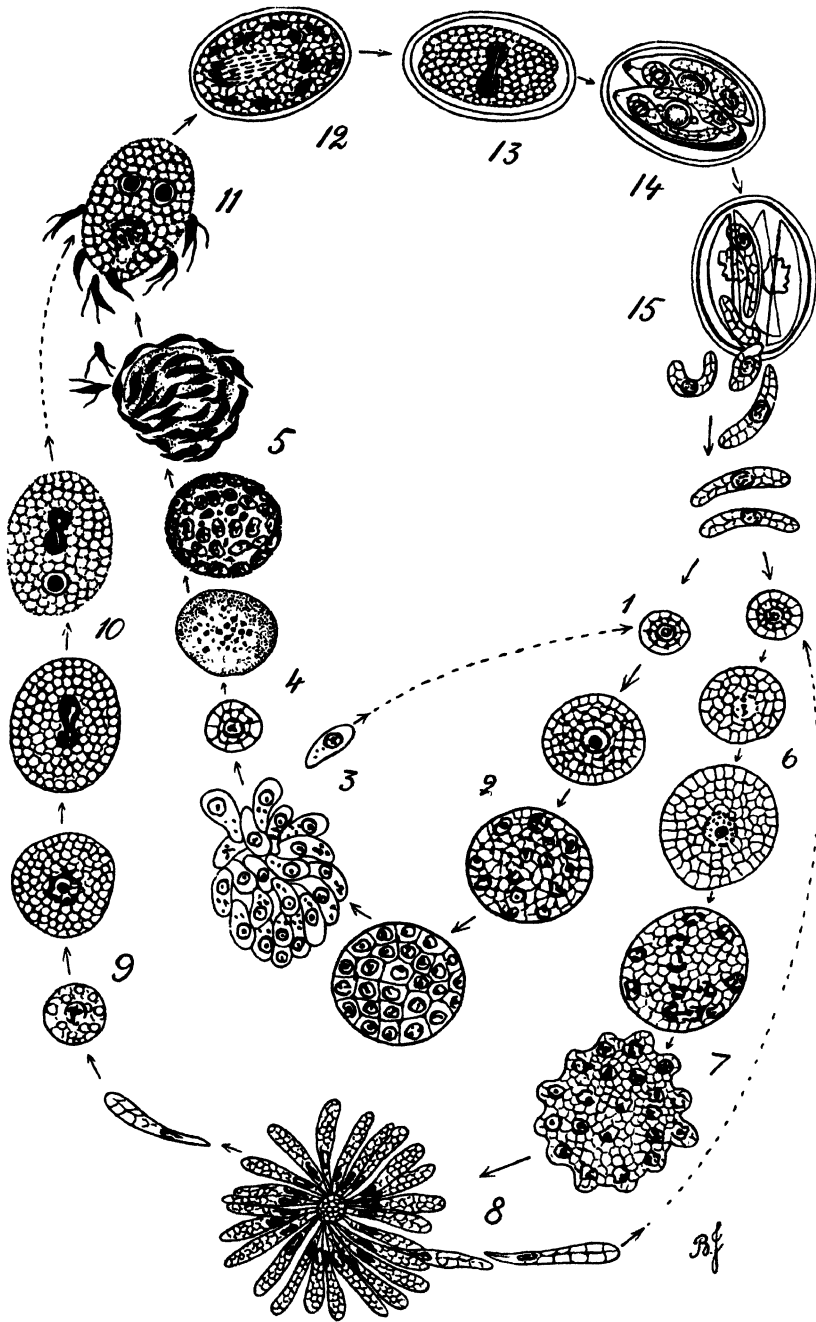 15. Escape of sporozoites.

FIG. 341.—LIFE-CYCLE OF *Cyclospora caryolytica*. (AFTER SCHAUDINN, 1902.)
(× *ca.* 1,400).

now approach the macrogamete, and frequently several of them enter the cytoplasm, though the nucleus of only one fuses with the macrogamete nucleus. The nuclei of the other microgametes are functionless, and eventually are cast off in the residual body. After fertilization the oöcyst is formed around the zygote, which now passes out of the intestine in the fæces. On the ground the further development takes place, and two sporoblasts are produced, which become encysted in bivalved sporocysts, like those of *E. schubergi*. Within the sporocyst each sporoblast produces two sporozoites and a residual body consisting of the unused cytoplasm, in which are the bulk of the surplus food material, the remains of the degenerating maturation nuclei, and the nuclei of the superfluous micro-gametes (Fig. 341, 11-14). The ripe oöcysts remain on the ground without further change till they are casually eaten by a mole, when the sporozoites escape and invade the gut epithelium.

The above description is that given by Schaudinn, but Reichenow (1921*a*) has expressed some scepticism as to the accuracy of his account. He believes that the two lines of schizogony may represent a double infection with two different coccidia, or that the two types of schizogony merely represent variations in the ordinary schizogony process, which is by no means constant in many other forms. The final schizogony, which gives rise to merozoites which are in reality young gametocytes, since they grow into these, may differ, in the characters of the schizont and number of merozoites produced, from that which has occurred earlier on, and which gave rise to merozoites destined to become schizonts again. From what has since been discovered in coccidia, the process of matu-ration of the macrogametocyte, as described by Schaudinn, requires reinvestigation.

Phisalix (1923, 1924) gives the name *C. viperæ* to a form which occurs in the intestine of the viper (*Vipera aspic*). The oöcysts measure 16·8 by 12·6 microns. Each contains two sporocysts 10·5 by 8·4 microns in size. Schizogony, which occurs in the intestinal epithelium, gives rise to from four to eighteen merozoites according to the size of the schizonts. Phisalix (1924*a*, 1924*b*) describes two other species. *C. babaulti* occurs in *V. berus* and *C. tropidonoti* in *Tropidonotus natrix*.

(2) *Sub-Family* : ISOSPORINÆ.

This sub-family includes the single genus, *Isospora*, founded by Aimé Schneider (1881) for a parasite called by him *I. rara*, which he found in an unnamed black slug. His description and figures of the oöcyst were imperfect, but there appears to be sufficient indication that it contained two sporocysts, each of which had four sporo-zoites. This parasite has not been reinvestigated, and till this is done it is impossible to be quite certain about these characters. Labbé (1893) regarded the oöcysts of Schneider's parasite as containing two sporocysts, each with many sporozoites, and

established the genus *Diplospora* for a parasite which he had discovered in birds, the oöcyst of which definitely included two sporocysts, each with four sporozoites. Most authorities, including Laveran and Mesnil (1902c), regard Labbé's *Diplospora* as a synonym of *Isospora*. Another parasite which belongs to this genus is one which occurs in the kidney and intestine of the frog, which was discovered by Lieberkühn (1854). It was rediscovered and named *Klossia lieberkühni* by Labbé (1894), and later transferred by him (1896) to a new genus, *Hyaloklossia*, which Laveran and Mesnil (1902c) pointed out was a synonym of *Isospora*. The latter observers accordingly named the frog coccidium *I. lieberkühni*. Rivolta (1878), however, gave the name *Cytospermium ranæ* to a coccidium which Eimer (1870) had seen in the frog. Dobell (1919a), assuming that this is the form first seen by Lieberkühn, and later named by Labbé, thinks it possible that Rivolta's name has priority over Labbé's, and that the frog, *Isospora*, should be known as *I. ranæ*. It appears, however, that Rivolta, in naming the parasite, was referring to the form seen by Eimer, which was apparently found in the intestine and not in the kidney. From Eimer's description and figures it is impossible to tell whether he was dealing with an *Isospora* or an *Eimeria*, and as coccidia of both these genera occur in the intestine of frogs, Rivolta's name may apply to either of these. Eimer himself, who mentions Lieberkühn's parasite, did not appear to regard it as the same as the one he had found and figured. Hence Rivolta's name, *C. ranæ*, cannot be regarded as applying to Lieberkühn's parasite, the correct name for which is *I. lieberkühni*. Another point which arises from Rivolta's name is the possibility of his generic title, *Cytospermium* (1878), having priority over *Isospora* (1881). In this genus Rivolta included several distinct parasites. The first name used by him is *C. viride*. It was given to certain cyst-like bodies, which he states were found by Paulicki (1872) in the lungs of monkeys, and by Piana (1876) in the mesentery of fowls and the lungs of a monkey. It seems impossible to identify these structures with any known organism. The second species is *C. zurnii*, which is undoubtedly the *Eimeria* of cattle first seen by Zürn (1878). The third species is *C. ranæ*, which, as pointed out above, may be either an *Isospora* or an *Eimeria ;* while the fourth is *C. hominis*, a name given to a supposed coccidium of man seen by Eimer (1870). Here, again, it is impossible to be sure that the structures seen by Eimer were coccidia, and there are no grounds for assuming that they are identical with the undoubted coccidia (*Isospora*) seen by Kjellberg in man and recorded by Virchow (1860a). Rivolta's name, *C. hominis*, is thus a *nomen nudum*.

The fifth species of Rivolta is *C. villorum intestinalium canis*, an undoubted *Isospora* of the dog; while the sixth is *C. hepatis canis familiaris*, a name which refers unquestionably to the eggs of a Trematode seen by Perroncito in the liver of a dog. The name *Cytospermium* was thus given to a variety of different organisms. It was first given to questionable structures in the lungs of monkeys, and secondly to an undoubted *Eimeria*, so that it seems impossible to regard the name as having priority over *Isospora*. The name *Isospora* of Aimé Schneider (1881) can safely be taken at present as the correct one for those coccidia which have oöcysts containing two sporocysts, each of which has four sporozoites. The type species is *I. rara*, A. Schneider, 1881, of the black slug. If reinvestigation of this parasite should prove that its oöcyst does not contain two sporocysts, each with four sporozoites, then Labbé's name, *Diplospora*, with *D. lacazei* Labbé, 1893, a parasite of birds, as the type, will be the correct one.

Henry and Leblois (1925, 1926) have attempted to subdivide the genus *Isospora*. They conclude that two genera are represented—viz., *Isospora*, in which the sporocysts are pyriform in shape; and a new genus, *Lucertina*, in which the sporocysts are spherical or ellipsoidal. It is doubtful if these characters are of generic value.

The sub-family, as indicated above, contains the single genus *Isospora* Aimé Schneider, 1875, the best-known species of which are those which occur in cats and dogs, birds and frogs. Cats and dogs harbour three distinct species, while human beings are liable to infection with one, or probably two, species.

ISOSPORA IN CATS AND DOGS.

Cats and dogs are very commonly infected with species of *Isospora*. It has generally been supposed that only one species, *I. bigemina* (Stiles, 1891) occurred in these carnivores, but the writer (1923a), from observations made on these animals in England, found that three distinct species,
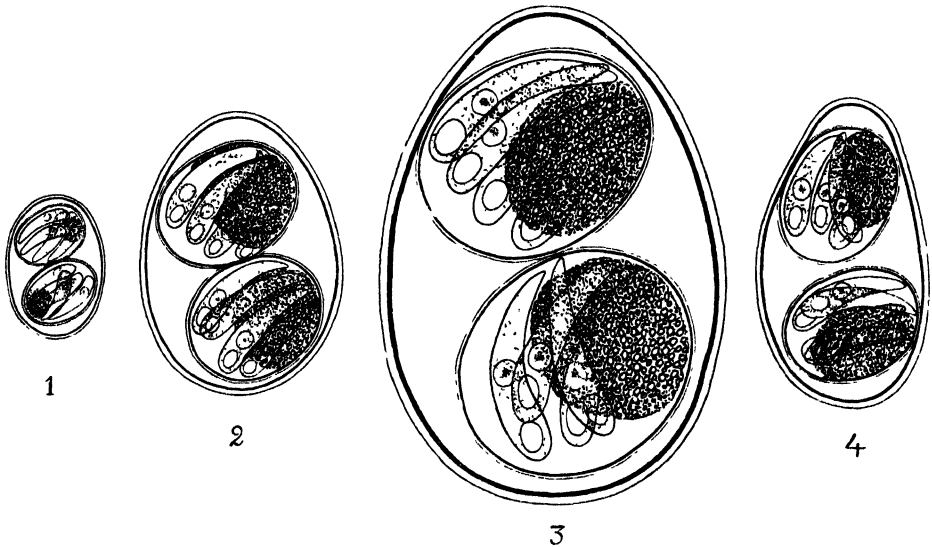

FIG. 342.—DIAGRAM OF OÖCYSTS OF *Isospora* OF CATS, DOGS, AND MAN (× *ca.* 1,400). (AFTER WENYON, 1923.)

1. Oöcyst of the small form which occurs in the deeper tissues of the villi of cats and dogs and man (*Isospora bigemina* and *Isospora hominis*)
2. Oöcyst of the intermediate sized form which occurs in the epithelium of the villi of cats and dogs (*Isospora rivolta*)
3. Oöcyst of the large form which occurs in the epithelium of the villi of cats and dogs (*Isospora felis*).
4. Oöcyst of the large form which probably occurs in the epithelium of the villi of man (*Isospora belli*).

all of which had previously been recorded in the literature, had been grouped under this one name (Fig. 342). The three species (*I. bigemina*, *I. rivolta*, *I. felis*) differ in the size of the oöcysts and sporocysts; also as regards the localization of the infection in the tissues of the villi. The writer (1923a) found that *I. rivolta* and *I. felis* reproduced only in the epithelium, while *I. bigemina* occurred in the subepithelial tissues, where

the oöcysts reached maturity. The writer and Sheather (1925) have since noted that in the acute phase of an infection in the dog *I. bigemina* reproduces in the epithelium like the other forms, and that immature oöcysts are passed in the fæces. In the chronic phases of an infection in both the cat and the dog, the parasite appears to be limited to the sub-epithelial tissues. *I. rivolta* also may reproduce in the subepithelial tissues.

The first of the parasites to receive a name was the one of intermediate size seen by Grassi (1879). He gave it the name *Coccidium rivolta*. Stiles (1891) proposed the name *C. bigeminum* for the small one he found in the dog, while the writer (1923a) gave the name *I. felis* to the largest one. Practically all observers have assumed that only one species existed. Perroncito (1882) and Railliet (1895) appear to have realized that the small form differed from that of intermediate size seen by Grassi. Pospiech (1919), investigating the oöcysts of these parasites in Germany, came to the conclusion that there were actually four species, three of which corresponded with the three forms described above, while one had oöcysts intermediate in size between those of *I. bigemina* and *I. rivolta*. Davis and Reich (1924) have noted that the three forms occur in cats and dogs in California. They also found sporocysts of a form which occupied an intermediate position between those of *I. bigemina* and *I. rivolta*. Nieschulz (1925) has also seen these in a young dog in Holland. The oöcysts measured 14 to 17 by 8 to 10 microns, and he suggests that they may belong to a distinct species. The whole question of the three species was discussed by the writer (1923a), but there appears to have been some confusion regarding the dimensions of the oöcysts and sporocysts, while further information has recently been obtained.

Isospora bigemina (S iles, 1891).—This coccidium, which is an intestinal parasite of dogs, cats, and polecats, was first described by Finck (1854), who gave an accurate description of the oöcysts with their two sporocysts. He referred to them as *corpuscles géminés*, but did not, however, see the sporozoites within the sporocysts. He came to the conclusion that the bodies were products of fat absorption. They were next seen by Virchow (1860) in the dog, and by Leuckart (1860) in the same animal. Rivolta (1874, 1877, 1877a, 1878) gave a fairly good description of the oöcysts with their paired sporocysts, including four sporozoites and residual body. He called the parasite *Cytospermium villorum intestinalium canis*. The parasite was next seen in the dog by Railliet and Lucet (1888), who later (1890, 1891) found it in dogs, cats, and polecats (*Putorius putorius*). Stiles (1891) proposed the name *Coccidium bigeminum* for the form in the dog, while Railliet and Lucet (1891) distinguished three varieties—viz., *C. bigeminum* vars. *canis, cati,* and *putorii* in the dog, cat, and polecat respectively. These differ from one another in the size of the sporocysts. Stiles (1892) gave a further description with figures of the form in the dog. All these authors are in accord as regards the dimensions of the sporocysts, that they occur in the deeper tissues of the villi, and that their development is completed in the body. From 1892 onwards no one appears to have seen this parasite till the

writer (1923*a*) discovered it in a cat in London, and found that the previous descriptions were remarkably accurate, and that it was undoubtedly distinct from the larger forms of *Isospora* which occur in these animals. The writer and Sheather (1925) have now found that during the acute phases of an infection in the dog reproduction takes place in the epithelium, that immature oöcysts are passed in the fæces and that they attain maturity outside the body. It appears that in the chronic phases of infection the oöcysts occur in the subepithelial tissues, and develop to maturity in this situation.

All observers who had studied *I. bigemina* in cats and dogs were in agreement as to the presence of mature oöcysts in the subepithelial tissues and the absence of parasites from the epithelium (Figs. 343, 344). The
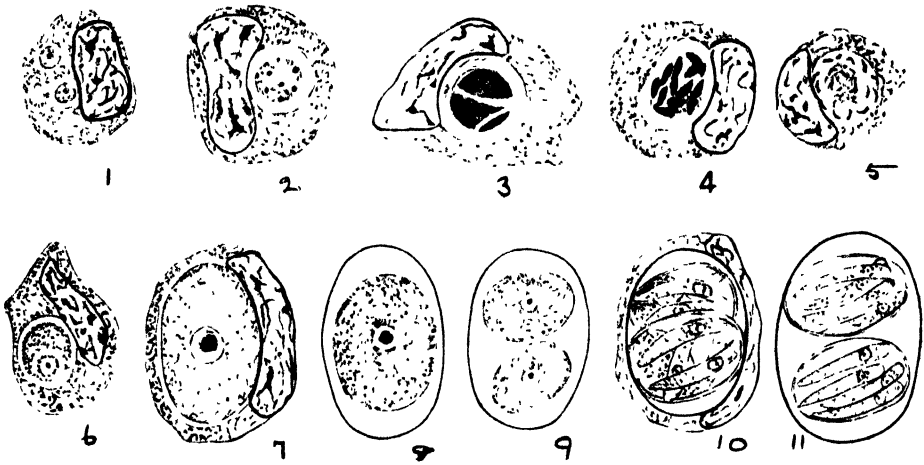

FIG. 343.—*Isospora bigemina* FROM SECTIONS OF THE SMALL INTESTINE OF THE CAT (× 2,000). (AFTER WENYON, 1923.)

1-4. Schizogony   5 Possible microgametocyte with microgametes.
6-7. Growth of macrogametocyte.   8-11 Development of oöcyst in tissues of villi.

writer (1923*a*), having observed the same distribution of the parasite, concluded that its entire development took place in the subepithelial tissue. Recently, however, the writer and Sheather (1925) have had an opportunity of studying the intestine of a dog which had been killed during the acute phase of infection. It was found that the whole of the epithelium of the small intestine from the stomach downwards was crowded with reproducing parasites. These occurred in the epithelial cells on the lumen side of their nuclei, while there appeared to be no tendency towards invasion of the subepithelial tissues. The schizonts were small, and had a diameter when mature of not more than 5 microns. Each gave rise to eight minute merozoites. In addition, there were numerous macrogametocytes with a diameter of about 7·5 microns. During life this dog

passed in its fæces numerous immature oöcysts measuring 10 to 14 by 7·5 to 9 microns, the sporocysts measuring 7·5 to 9 by 5 to 7 microns. In another puppy studied by the writer large numbers of oöcysts of the same dimensions were passed during the course of three or four weeks, during which the animal suffered from diarrhœa. The infection gradually subsided, and eventually disappeared. The oöcysts completed their development outside the body in the usual manner, forming two sporocysts, each
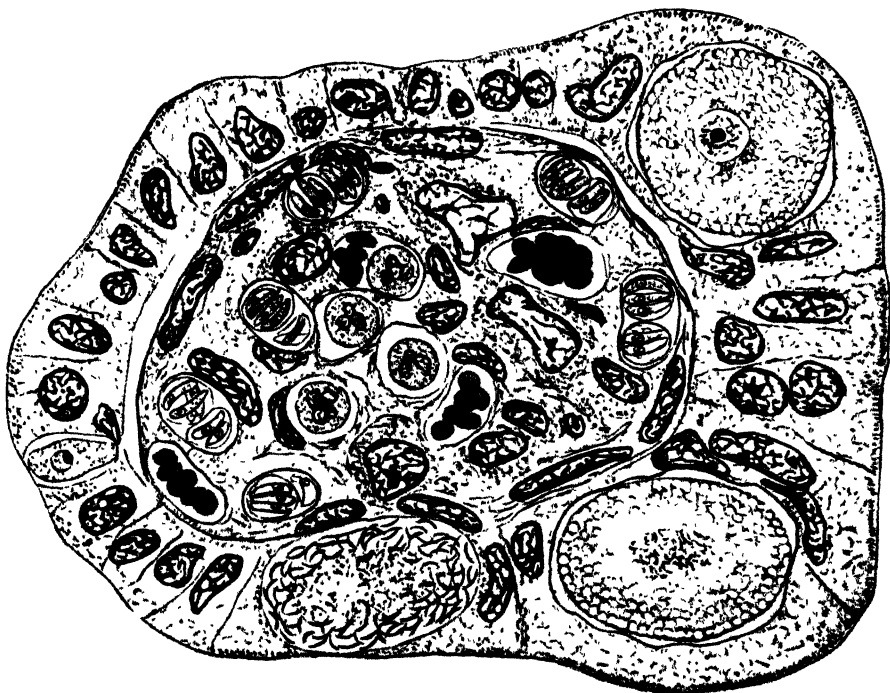

Fig. 344. Section of Villus of Cat, showing *Isospora felis* in the Epithelium, and *I. bigemina* in the Subepithelial Tissues (× 1,000). (After Wenyon, 1923.)

In the epithelium are seen two mature macrogametocytes, one partially grown macrogametocyte, and a microgametocyte with microgametes. In the tissues are six mature oöcysts of *Isospora bigemina*.

with four sporozoites and a residual body. They resembled the oöcysts previously described by the writer (1923a) as occurring in the subepithelial tissues of the villi of a kitten, except that in this situation the oöcyst wall was thinner and more easily ruptured. In addition to the oöcysts, a small number of schizonts, macro- and micro-gametocytes were found in the subepithelial tissues of the kitten (Fig. 343).

The writer has examined portions of the small intestine of a number of dogs collected for him by Mr. Burgess of Colombo. Oöcysts of *I. bigemina*

were discovered in six dogs. In each instance they were in the sub-epithelial tissues, were fully developed, and measured 18 to 20 by 14 to 16 microns. The sporocysts measured 13·5 to 15·5 by 9 to 10 microns. Sheather (1923) has observed immature oöcysts of what may be this parasite in the fæces of a dog in England.

It thus appears that in the acute stages of infection with *I. bigemina* reproduction occurs in the epithelium, and immature oöcysts are passed in the fæces, while in the chronic stages the oöcysts become mature in the subepithelial tissues. Furthermore, there appear to be oöcysts of two types. The smaller ones, which measure 10 to 14 by 7·5 to 9 microns, occur in both cats and dogs, while the larger ones, measuring 18 to 20 by 14 to 16 microns, have hitherto been seen only in dogs. The writer attempted to infect four kittens with the small oöcysts passed by the puppy mentioned above, but, though enormous numbers of fully developed oöcysts were swallowed by the kittens, no infection occurred. It hardly seems probable that the immature oöcysts passed in the fæces in the acute infection belong to species distinct from those which give rise to mature oöcysts in the subepithelial tissues in chronic infections, though the wall of the oöcyst in the case of the latter is thinner, more easily distorted and ruptured, and often closely wrapped round the sporocysts. It is, neverthe-less, possible that the large and small oöcysts belong to different species. Railliet and Lucet (1891) recognized this, and believed that in the dog, cat, and polecat there occurred three varieties, which differed in the dimensions of the sporocysts (erroneously termed oöcysts by the writer, 1923*a*), which they give as follows:

I. bigemina var. *canis* : 12 to 15 by 7 to 9 microns.
I. bigemina var. *cati* : 8 to 10 by 7 to 9 microns.
I. bigemina var. *putorii* : 8 to 12 by 6 to 8 microns.

In connection with the larger forms of *I. bigemina* it has to be remem-bered that the oöcysts have only been seen in the subepithelial tissues, where the oöcyst wall is thin and closely wrapped round the mature sporocysts. The dimensions of the latter (13·5 to 15·5 by 9 to 10 microns) agree with those of the sporocysts of *I. rivolta*, though the oöcyst of this coccidium, which has only been seen in the fæces, is larger—20 to 25 microns in length in place of 18 to 20 microns. The writer and Sheather (1925) have noted that schizonts and male and female gametocytes of *I. rivolta* may occur in the subepithelial tissues, though this parasite usually reproduces in the epithelium. It is possible that in chronic infections mature oöcysts may be produced in the subepithelial tissues, and in this situation they may be smaller than when developed in the epithelium and passed undeveloped in the fæces. In this case the large

form of *I. bigemina* may in reality be *I. rivolta*, the oöcysts of which have reached maturity in the subepithelial tissues. The similarity in the size of the sporocysts is in favour of this view.

As noted above, the writer (1923a) found that the tissues of the villi in the case of a chronic infection in a kitten were infiltrated with oöcysts in all stages of development (Fig. 344). Enormous numbers were present in some villi, which were swollen and evidently altered in appearance. Though the fæces of this cat had been examined on several occasions, neither the oöcysts nor the sporocysts of *I. bigemina* were discovered,

though the undeveloped oöcysts of the large *I. felis* were constantly present. It would seem that, in the case of chronic infections with *I. bigemina*, the oöcysts do not leave the villi regularly. They probably escape only when a damaged villus actually breaks down. It results that infection with this coccidium would be frequently overlooked if fæcal examinations alone were made. The infection in the cat was only discovered by the writer when scrapings from the wall of the small intestine were made after death. The infection

Fig. 345.—Oöcysts of *Isospora felis* and *I. rivolta* (× 230). (Original.)
Concentration from fæces of cat by Sheather's method

in the Colombo dogs was of a similar type, though less intense. Adler informs the writer that kittens in Palestine are commonly infected with *I. bigemina*.

It is evident that the true relationship of the various types of *I. bigemina* can only be determined by a series of carefully conducted cross-infection experiments.

Isospora rivolta (Grassi, 1879).—This coccidium, which was first seen by Grassi (1879) in the fæces of cats, has an oöcyst about 20 to 25 microns in length. It was also seen by Wasielewski (1904) in these animals, together with the large *I. felis*. He also observed it in dogs. Reichenow (1921a) described and figured the oöcyst from dogs in Germany, while Brown and Stammers (1922) and the writer (1923a) found it in the fæces of
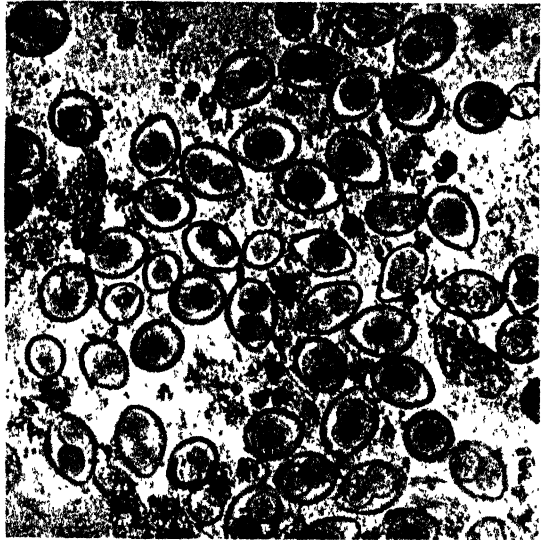
dogs in England. The writer has recently seen it in a cat, together with *I. felis* (Fig. 345). Grassi (1879, 1881*a*) noted that it developed in the intestinal epithelium, while Zapfe (1923) has given some details of the asexual reproduction which occurs in this situation. It resembles that of *I. felis*, but the various stages are correspondingly smaller in size. The oöcyst varies in length from 20 to 25 microns and in breadth from 15 to 20 microns (Figs. 346 and 350, 5). It is distinctly ovoid in shape, while one end may be more pointed than the other. At the pointed end a micropyle can sometimes be distinguished. A small residual body may be present in the oöcyst. The sporocysts are elongated bodies with rounded ends, and measure about 16 by 10 microns. Each contains four sporozoites and a large residual body filled with globules of a refractile material. As noted above, the writer and Sheather (1925) have noted the fact that *I. rivolta* may reproduce in the subepithelial tissues, and produce male and female gametocytes in this situation. The possibility of oöcysts
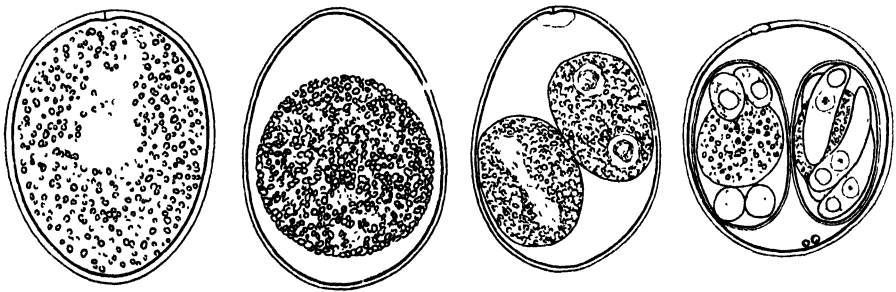

FIG. 346.—*Isospora rivolta* : STAGES IN DEVELOPMENT OF OÖCYST OUTSIDE THE BODY OF THE DOG (× 1,500). (AFTER WENYON, 1923.)

becoming mature in the subepithelial tissue and of their resemblance to the large forms of *I. bigemina* has already been considered (p. 812).

Adler (1924) has described as *I. viverræ* a parasite of the civet cat (*Viverra civetta*) of West Africa. It is very similar to *I. rivolta*, but as cats and dogs fed on heavily infected material failed to become infected, it was regarded as a distinct species. It is necessary, however, to be cautious in drawing conclusions from such experiments. Recently the writer obtained large numbers of ripe oöcysts of *I. bigemina* from a dog, and failed entirely to infect four kittens and a puppy. Adler informs the writer that *I. rivolta* is very common in kittens in Palestine.

Isospora felis Wenyon, 1923.—This parasite was first described by Wasielewski (1904) as *Diplospora bigemina*. He gave a complete account of the development of the oöcyst, and also noted free motile merozoites in the intestinal contents and schizogony stages in the epithelium. It was later studied in detail by Swellengrebel (1914*a*), who described the main

features of its life-cycle. Swellengrebel described the process of schizogony, the later stages of the development of the microgametocyte and formation of microgametes, the development of the macrogametocyte and oöcyst. He also described a curious process of parthenogenesis of the macrogametocyte. It is clear that this was the result of misinterpretation of certain appearances in sections of macrogametocytes. Swellengrebel noted that the parasite was confined to the epithelium of the villi of the small intestine. Hall (1917) observed the parasite in dogs in Detroit, and came to the conclusion that it was not the small *I. bigemina* described by Stiles, but Hall and Wigdor (1918) concluded that it was a larger form of this parasite. The oöcysts of *I. felis* were seen by the writer and O'Connor (1917) in cats in Alexandria, and by Dobell (1919a) in cats in England. Dobell, with previous observers, regarded it as *I. bigemina*, though he pointed out that Grassi's name, *I. rivolta*, which, however, refers to a distinct parasite, had been given to the coccidium before Stiles's name, *I. bigemina*. Dobell and O'Connor (1921) definitely referred to this parasite as *I. rivoltæ*, a name which had previously been used by Leuckart (1886). Dobell and O'Connor still regarded all these coccidia of cats and dogs as belonging to one species, though Dobell (1919a) suggested that the form in the dog might be distinct from that in the cat. Reichenow (1921a) definitely asserted that the form in the dog was smaller than that in the cat, and was probably a distinct species. Nöller (1921), without giving any details, writes of a small and a large form in the cat. Marotel (1921) again referred to the large form in the cat, and proposed to name it *I. cati*, a name, however, which was previously given by Raillet and Lucet (1891) to the variety of *I. bigemina* of the cat (*I. bigemina* var. *cati*). The writer (1923a) proposed the name *I. felis* for this parasite, which is very common in young cats in England. It does not as a rule, affect the kittens to any extent, but they often suffer from diarrhœa, which is probably the result of infection with this coccidium. It has been reported from a dog in England by Sheather (1923).

The developmental stages occur in the epithelial cells of the villi of the small intestine and also the cæcum (Figs. 344, 347). They are peculiar in that the schizonts, macrogametocytes, and microgametocytes retain their elongate gregariniform character to a comparatively late stage of growth. They are often attached to the surface of the vacuole, in which they lie, by their more pointed anterior ends, where a kind of sucker may occur. The smallest asexual forms are about 5 microns in length. These grow into ovoid schizonts, having a length of about 12 microns and containing eight nuclei, the result of repeated mitotic divisions of the original nucleus. Usually eight merozoites are produced (Fig. 347, 1-9). Occasionally, however, as many as sixteen or more are formed. The micro-
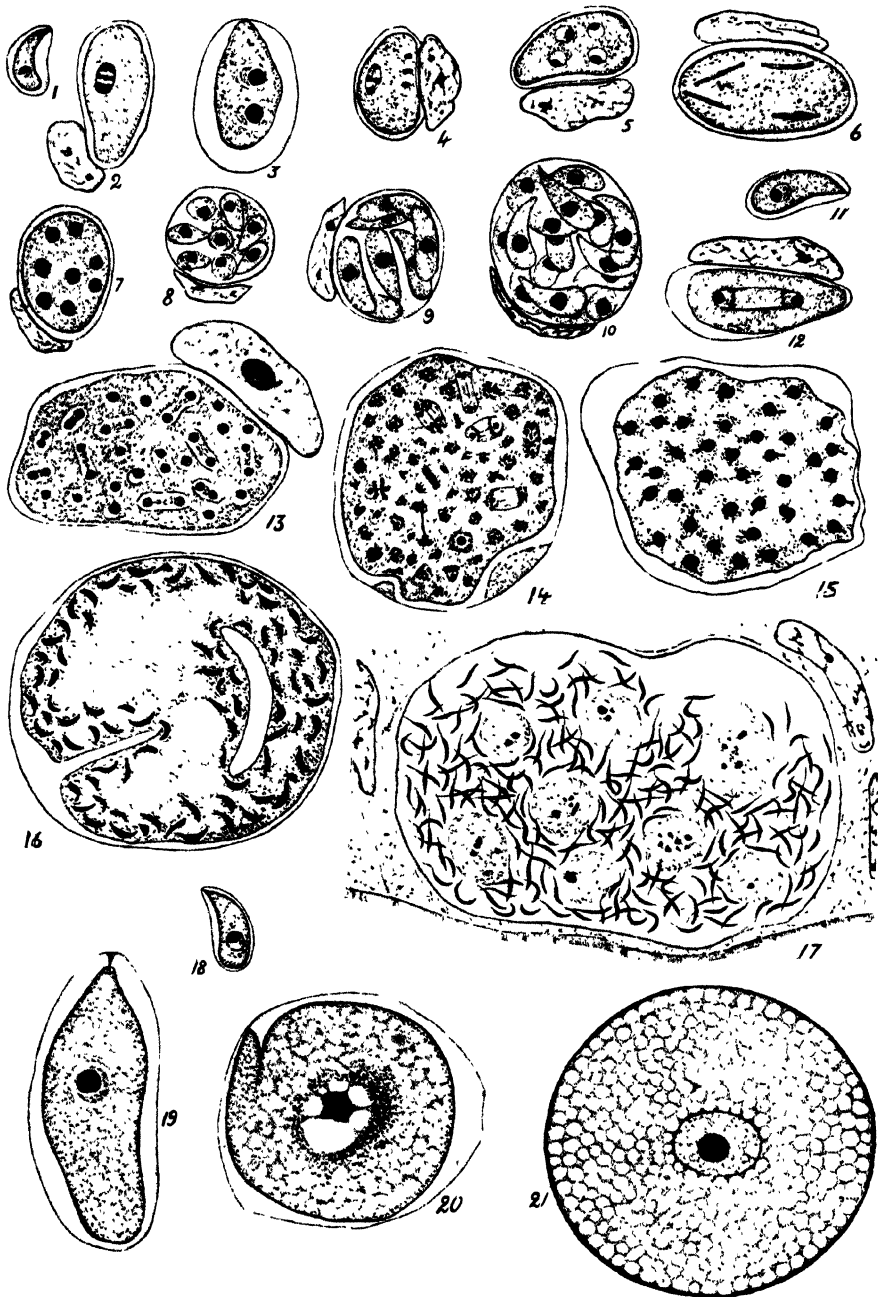
FIG. 347.—*Isospora felis:* DEVELOPMENT IN THE INTESTINAL EPITHELIUM OF
THE CAT (× 2,000). (AFTER WENYON, 1923.)

gametocyte commences its growth as a merozoite, and increases in size till it measures about 20 by 30 microns or more. By repeated mitotic divisions of the nucleus which occur during the growth an enormous number of nuclei are formed. Finally, the nuclei become arranged on the surface of the parasite, as also on the surface of vacuoles and fissures or folds which have formed. Here each becomes concentrated to form a solid mass of chromatin, from which a finger-like process grows out. The whole mass then becomes sickle-shaped, and finally an elongate microgamete, which, according to Swellengrebel (1914a) has two flagella attached to its blunt anterior end, is formed. A single microgametocyte may form over 2,000 microgametes (Fig. 347, 11-17). The macrogametocyte, commencing as a merozoite, becomes a large ovoid cell varying in length from 25 to 35 microns. During its growth the cytoplasm changes in character. Like the early stages of growth of the schizont and microgametocyte, the macrogametocyte at first stains very deeply. Later it has less affinity for stains, and there appears around the nucleus a number of deeply staining granules, while irregularly shaped masses of a deeply staining substance appear in the cytoplasm. After this there are formed in the cytoplasm a number of globules of a refractile substance, which finally fill the entire macrogametocyte. Concurrently with this change, the deeply staining substance disappears. As in the nuclei of the schizonts and microgametocytes, the karyosome is present at all stages, and there is no indication that it is thrown out of the nucleus of the macrogametocyte, or that it disintegrates before fertilization takes place, for it is still present in the zygote (Fig. 347, 18-21). The wall of the oöcyst is formed round the macrogametocyte as a thin membrane while it is in the epithelium, but it does not become the characteristic resistant structure till the oöcyst falls into the lumen of the intestine. Fertilization has not actually been observed, but this probably takes place after the oöcyst has formed. As in the case of *I. rivolta*, the zygote does not commence to divide till the oöcyst has left the body in the fæces (Fig. 348). Its development was described by Wasielewsky (1904), who gives the measurements of the oöcysts as 22 to 40 by 19 to 32 microns, but it is probable that he had a mixed infection of *I. felis* and *I. rivolta*. The writer (1923a) found the oöcysts of *I. felis* measured 39 to 48 by 26 to 37 microns. Occasionally shorter or even longer forms occur. Dobell (1926a) states that he has seen oöcysts only 30 microns in length. The first stage in development of the oöcyst, which according to Dobell may occur in the intestinal epithelium, is the retraction of the zygote to form a spherical mass 18 to 25 microns in

1-8. Schizogony.   9. Schizont producing eight large merozoites.
10. Schizont producing sixteen merozoites.
11-17. Growth of microgametocyte and production of macrogametes.
18-21. Growth of macrogametocyte.

diameter. This divides to form two sporoblasts 16 to 18 microns in diameter. There may be a small residual body. The sporoblasts elongate and form thick-walled sporocysts measuring 20 to 27 by 18 to 21 microns, within each of which are formed four sporozoites and a large residual body. The sporozoites are more rounded at one end than the other. They have a central nucleus with a karyosome, while near the blunt end is a large ovoid refractile body.

Weidman (1915) described a parasite of " swift foxes " in America, the oöcysts of which resemble those of *I. felis*. Believing that all these coccidia of carnivores belonged to the species *I. bigemina*, he suggested that it was a variety *canivecolis*. Mesnil (1916) and Hall and Wigdor (1918) refer to this parasite as *Coccidium bigeminum canivecolis*. If it is
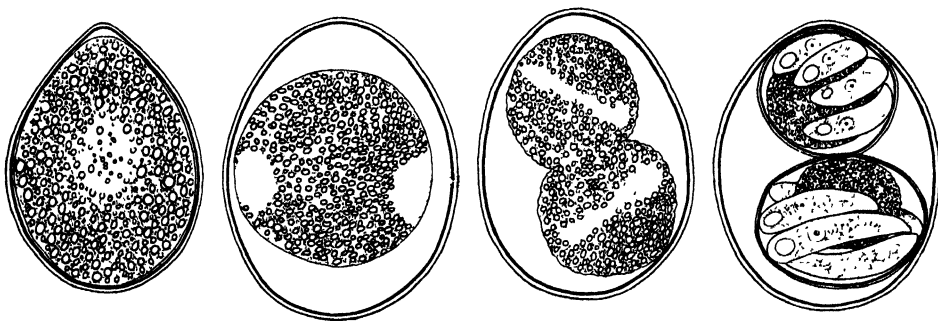
FIG. 348.—*Isospora felis* : STAGES IN DEVELOPMENT OF OÖCYST OUTSIDE THE BODY OF THE CAT (× 740). (AFTER WENYON, 1923.)

distinct from *I. felis*, its name will be *I. canivecolis*, as suggested by the writer (1923a). Möller (1923) has noted the oöcysts of a species of *Isospora* in the fæces of a lion in the Zoological Gardens in Berlin. They bore a close resemblance to those of *I. felis*.

As regards the distribution of the three species of *Isospora* in cats and dogs, it appears that they may occur in both these animals. Grassi (1879, 1881a) noted only *I. rivolta* in the cat. Wasielewski (1904) and the writer have seen both this form and *I. felis* in this animal and *I. rivolta* in dog. Reichenow (1921a) in Germany found *I. felis* in cats and *I. rivolta* in dogs. Sheather (1923) and the writer and Sheather (1925) have seen both *I. felis* and *I. rivolta* in the dog. Swellengrebel (1914a) and Marotel (1921) saw *I. felis* in cats, and Hall and Wigdor (1918) both this form and *I. rivolta* in dogs in Detroit. Similarly, *I. bigemina* occurs in both cats and dogs. There is as yet no experimental evidence that the forms in cats are distinct from those in dogs, apart from the negative result obtained by the writer in attempting to infect kittens with *I. bigemina* from the dog.

The dimensions in microns of the oöcysts and sporocysts of the cat and dog parasites, as observed by the writer and Sheather, are as follows:

		Oöcysts.	Sporocysts.
I. felis ..	.. .. ..	39–48 × 26 –37	20–27 × 18–21
I. rivolta	.. ..	20–24 × 15 –20	12–15 × 9–10
I. bigemina (large)	.. ..	18–20 × 14 –16	13·5–15·5 × 9–10
I. bigemina (small)	.. ..	10–16 × 7·5–10	7·5–10 × 5–8

ISOSPORA IN MAN.

The first reference to an *Isospora* in man is that of Virchow (1860a), who recorded a case brought to his notice by Kjellberg. Virchow stated that psorosperms were present in the tissues of the villi of the small intestine, and that they agreed entirely with the forms he had seen in dogs. As noted above, the latter were oöcysts of *I. bigemina*, so that the only legitimate conclusion, as pointed out by the writer (1923a), is that Virchow saw in man a small *Isospora* like *I. bigemina* of cats and dogs. Eimer (1870) described certain bodies in the intestinal epithelium of two human beings which he examined post-mortem in Berlin. He stated that they resembled the psorosperms he had seen in mice and other animals. It is quite impossible to identify these bodies from Eimer's description. They may or may not have been coccidia, and, if they were, they certainly cannot be recognized as *Isospora*, for Eimer gives no clue as to the number of sporocysts or sporozoites. Rivolta (1878) gave the name *Cytospermium hominis* to the psorosperms described in man by Eimer, and as these cannot be identified, Rivolta's name is a *nomen nudum*. Railliet and Lucet (1890) described certain bodies which they saw in the fæces of a woman and her child in France. Their dimensions were similar to those of the oöcysts of *I. bigemina*, but as no development was noted they cannot be identified with the psorosperms described by Virchow. Railliet and Lucet (1891), referring to Virchow's psorosperm, and not to the bodies seen by themselves, and regarding it as a variety of the small *Isospora* of dogs, named the parasite *Coccidium bigeminum* var. *hominis*, so that the name for the human parasite becomes *I. hominis* (Railliet and Lucet, 1891). During the war the oöcysts of an *Isospora* were discovered in the fæces of man first by Woodcock (1915) and then by the writer (1915f). Since that time, according to Connal (1922), who has given a complete account of the records, over 150 cases of infection of this *Isospora* have been met with. The oöcysts of this form vary in length from 25 to 30 microns. Dobell (1919a) came to the conclusion that Virchow had probably seen the same parasite, but this view was based on the assumption that the various *Isospora* which occurred in cats and dogs, including the small one first seen by Finck, belonged to one species, *I. bigemina*. When once it is realized that the small form in the cat is distinct from the larger ones, it becomes evident that the one seen by Virchow in man, which he said was exactly like the small one in the dog and cat, cannot be identified with the much larger one discovered in man during the war. It seems evident, as noted by the writer (1923a), that Virchow saw in man a small *Isospora* like *I. bigemina* of cats and dogs. This one, which is *I. hominis* (Railliet and Lucet, 1891), has not since been rediscovered, with the possible doubtful exception of the bodies seen in the two cases recorded by Railliet and Lucet (1890). This seemed surprising, but the practice of examining fæces only, as has been the universal custom in recent years, is not calculated to reveal a parasite which is buried in the deeper tissues of the villi. As noted above, the writer discovered *I. bigemina* in the cat only when scrapings from the wall of the small intestine were made. Repeated examinations of the fæces had only revealed the oöcysts of *I. felis*, which develops in the epithelium. It seemed not improbable,

therefore, that the small *I. hominis* of man had not been rediscovered, because the practice of examining the wall of the small intestine at *post-mortem* had been largely given up. On the other hand, the larger oöcysts of the *Isospora* discovered during the war are present in the fæces, a fact which suggests that, like *I. rivolta* and *I. felis*, it is a parasite of the intestinal epithelium. The writer (1923*a*) suggested the name *I. belli* for this form. The conclusions arrived at by the writer (1923*a*, 1926) have recently been supported by Reichenow (1925), who has discovered in man the fully developed sporocysts of a species of *Isospora*.

Isospora hominis (Railliet and Lucet, 1901).—As already remarked, until recently there was only one authentic record of this parasite—namely, that of Virchow (1860*a*). His statements regarding it are very precise. He says the psorosperms, as he terms the sporocysts, occurred in the interior of the villi towards their tips, and that they agreed entirely with those he had seen in dogs, which again he identifies with the *corpuscles géminés* found by Finck (1854) in the cat. It appeared evident to the writer (1923*a*) that human beings are liable to infection with a small coccidium like *I. bigemina*. Re-

cently Reichenow (1925), examining the stool of a man suffering from amœbic dysentery, discovered two mature sporocysts of a species of *Isospora* (Fig. 349). The size was 16 by 10·5 microns. They are slightly larger than the sporocysts of the larger forms of *I. bigemina*, but in view of the fact that they were fully developed, Reichenow regards them as the sporocysts of
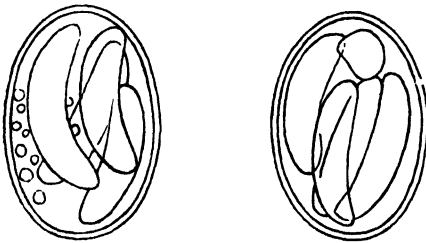
Fig. 349.—*Isospora hominis* (?): Mature Sporocysts from Human Fæces (× 2,000). (After Reichenow, 1925.)

I. hominis. It is probable, as noted by Dobell (1926*a*) and the writer (1926), that they are sporocysts of *I. belli*, with which they agree fairly closely, and that its oöcysts may reach maturity before leaving the body. It seems not improbable that if examinations of the wall of the small intestine are made at *post-mortem*, the small *Isospora hominis* will be rediscovered.

Isospora belli Wenyon, 1923.—As noted above, the immature oöcysts of this coccidium were discovered by Woodcock (1915) in the fæces of patients invalided from Gallipoli. He suggested that it belonged to the genus *Isospora*. The writer (1915*f*) demonstrated that this was actually the case by observing the complete development of the oöcyst. Since that time the oöcysts have been discovered by various workers in over one hundred and fifty cases from Egypt, Gallipoli, Salonika, Mesopotamia, Palestine, and other parts of the world. It is remarkable that in most instances the troops which were found infected during the war had been in contact with Turks, so that Turkey may be the endemic area of the

infection. Since the war cases have been recorded from other localities. The organism has only been studied in the oöcyst phase (Figs. 342 and 350, 7-10). It is probably a parasite of the epithelial cells of the small intestine, and it is in this situation that the schizogonic cycle and the development of the micro- and macro-gametocytes will very likely be found. So far, however, no *post-mortem* material has been obtained, and it can only be conjectured that the intestinal phases will be similar to those of *I. felis* of the cat. Several opportunities of obtaining fixed material from the intestines of cases which have died of other infections have unfortunately been missed.

The oöcysts, according to Dobell (1919*a*), vary in length from 25 to 33 microns and about half this in width. They are thus elongate bodies, and one end is rather more constricted than the other, giving the appearance of a kind of neck. Though the length is usually about twice the breadth, this is by no means constant, as broader forms may occur.

As found in the freshly passed human fæces, the oöcysts are transparent colourless bodies. The wall is composed of an outer thick layer showing a double contour and an inner and much finer membrane. At the narrower end there is an indication of a micropyle, against the inner side of which may be sometimes seen a small mass of granular material. Within the cyst is usually seen a spherical cytoplasmic body containing globules of a more refractile material. Sometimes the cytoplasmic body almost completely fills the oöcyst, and this is undoubtedly its condition when the oöcyst is first formed. The retraction of the enclosed cytoplasm to the spherical form is the first stage in its development. Sometimes, again, the freshly passed oöcyst will contain two masses of cytoplasm— the sporoblasts. If fæces containing oöcysts are spread out in a Petri dish and kept at the ordinary laboratory temperature of England, the oöcysts will complete their development in three to four days. In Egypt the writer and O'Connor (1917) found that twenty-four hours might suffice. The first stage is the shrinkage of the cytoplasm to form the spherical body. The latter then divides into two sporoblasts, which become somewhat elongate and form around themselves cyst walls, thus becoming sporocysts, which measure 12 to 14 by 7 to 9 microns. The sporoblast within each sporocyst then divides into four sporozoites and a large spherical residual body which contains the surplus food reserve material. The sporozoites themselves are elongate structures rounded at the anterior end and tapering at the posterior. The cytoplasm, which is finely granular, has a vacuole containing refractile material at the rounded end. The nucleus, which is difficult to detect, lies at the junction of the anterior and middle third of the sporozoite. From analogy with other coccidia, it may be presumed that the oöcyst is now ripe for the infection of a new
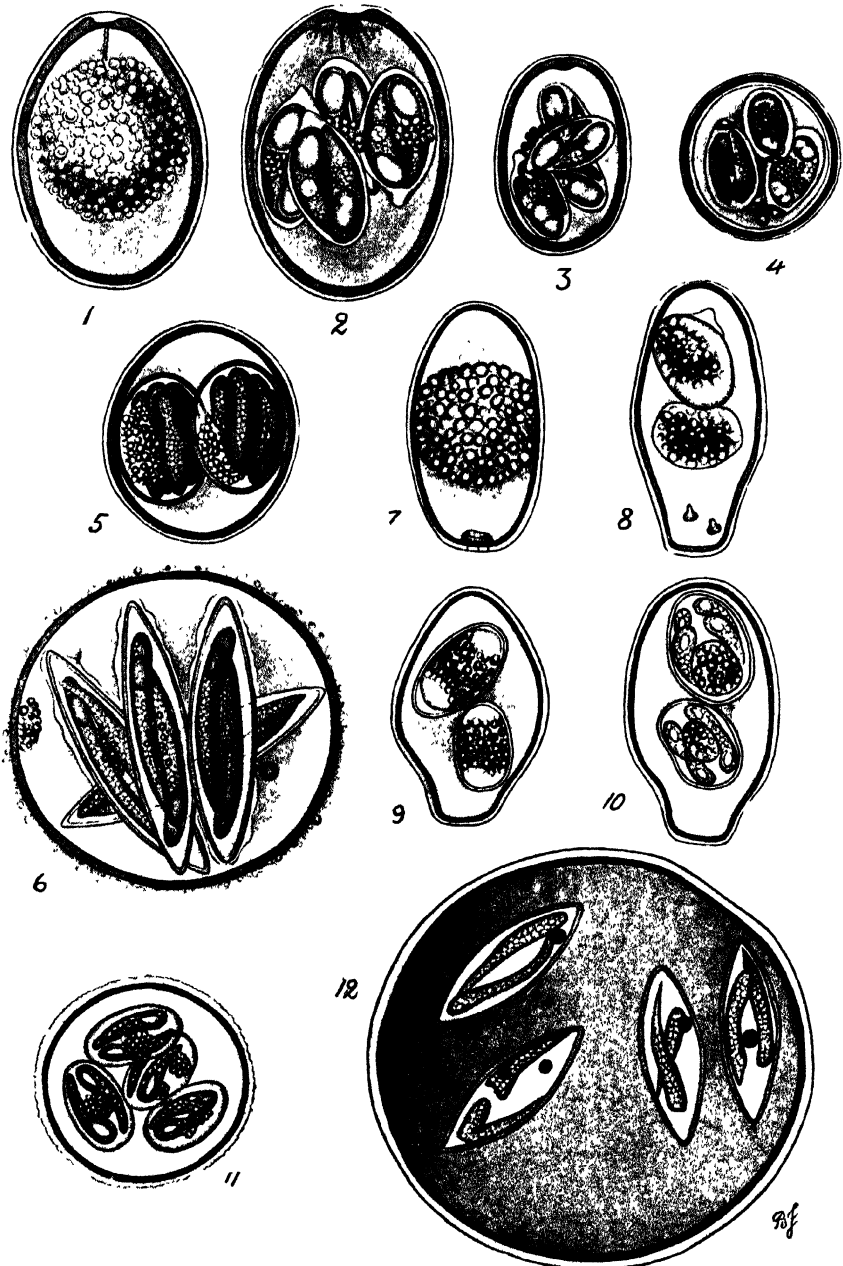
FIG. 350.—OÖCYSTS OF VARIOUS COCCIDIA FROM THE FÆCES OF MAN AND ANIMALS
(11, *ca.* 1,500; OTHERS, × *ca.* 1,300). (1-5, AFTER REICHENOW, 1921; 7-11,
AFTER WENYON, 1915; 6 AND 12, AFTER DOBELL, 1919, 1921; SLIGHTLY
MODIFIED.)

host. The writer and O'Connor (1917) noted that occasionally abnormal development might occur, a single sporocyst being secreted around the zygote within the oöcyst, and eight sporozoites and a residual body being formed within it. The development of the oöcyst has only been studied in fresh living material. The preparation of fixed and stained specimens presents difficulties owing to the highly impermeable nature of the oöcyst, which does not allow of the passage of fixatives.

The number of oöcysts present in the fæces is generally small. One or two to each cover-glass preparation, or even fewer, is the usual state of affairs, and as they are such highly transparent structures they are readily overlooked. Much larger infections have been occasionally observed by the writer and others. The oöcysts have not usually persisted in any case for more than a few days, but once in Egypt the writer and O'Connor (1917) noted their presence for twenty-five days.

As regards the pathogenicity of *I. belli*, the only reliable information is that given by Connal (1922), who observed a laboratory worker who had accidentally ingested material containing developed oöcysts. Six days later diarrhœa with abdominal discomfort set in. This persisted for four weeks, and oöcysts were discovered in the stools three weeks after the onset of symptoms, and were present more or less continuously for twelve days, after which they were not found. The stools had become normal again, and recovery was complete.

Attempts have been made to infect animals experimentally. The writer (1916) in England, and subsequently with O'Connor in Egypt (1917), attempted to infect kittens by feeding them with ripe oöcysts, but no infection occurred. Some of the kittens, however, showed infection with *I. felis*, which, however, has an oöcyst easily distinguished from that of *I. belli* on account of its larger size and more irregular shape. A mouse which was repeatedly fed also did not become infected. Later O'Connor (1919) attempted to infect two young puppies, but without result. Fantham (1917), however, claimed to have infected kittens, but it is not clear that *I. felis* was absolutely excluded, and, as Dobell (1919a) remarks, his statement that the infection in the kittens produced a " condition resembling that seen in the human intestine examined *post-mortem* " can hardly be accepted, since the changes produced in the human intestine have never been described.

The following records show the number of times *I. belli* has been found in cases examined: Woodcock and Penfold (1916), 10 in 384; Wenyon (1916),

1-2. *Eimeria stiedæ* from rabbit.
4. *Eimeria avium* from chicken.
6. *Eimeria oxyspora* (=*E. sardinæ*) from man.
11. *Eimeria wenyoni* (=*E. clupearum*) from man.
12. *Eimeria snijdersi* (=*E. sardinæ*) from man.

3. *Eimeria perforans* from rabbit.
5. *Isospora rivolta* from dog.
7-10. *Isospora belli* from man.

15 in 556; Roche (1917), 15 in 893; Savage and Young (1917), 6 in 1,088; Williamson (1917), 2 in 180; Martin, Kellaway and Williams (1918), 1 in 422; Cragg (1917), 4 in 613; Boney, Crossman and Boulenger (1918), 7 in 890; O'Connor (1919), 9 in 3,854. In addition to these, other cases have been recorded by various observers. Noc (1920) recorded a doubtful case from Saigon, and one which appears to be authentic from Dakar in West Africa. Kofoid, Kornhauser and Plate (1919), and Kofoid and Swezy (1920a), observed cases in America, not only amongst troops which had served abroad, but also amongst those of the home service. Haughwout (1921) has described a case seen by him in Manila. The infection may have been acquired in America. Porter (1918) recorded two cases amongst natives of South Africa, Brug (1922a) four indigenous cases from Java, and Connal and Young (1922) one from West Africa (Lagos). Connal (1922) gives an account of an infection of a laboratory worker who accidentally ingested oöcysts from this case. Newham and Robertson (1922) have noted three cases in Africa, two in Portuguese East Africa and one in Durban; while Boon van Ostade (1923) records a case from Malay, and Wassell (1923) one, hardly recognizable from his drawings, from China. Knowles (1924) has observed two cases in Calcutta, Castex and Greenway (1923) one in the Argentine. Pinto and Pacheco (1925) one in Brazil, and Pons (1925) two in Saigon.

ISOSPORA IN OTHER MAMMALS.

Apart from the species of *Isospora* described above, mammals appear to be particularly free from coccidia of this genus. Apparently the only form which has been noted is one which was briefly described and figured by Virchow (1860a) as occurring in the kidney of a bat. No one has rediscovered this parasite.

ISOSPORA IN BIRDS.

Coccidia belonging to this genus are fairly common parasites of birds, such as the sparrow, crow, and many passerines. They were probably first seen by Eimer (1870) in the sparrow. Rivolta (1869) saw coccidia in fowls and other birds, but though in the form from the bird he noted the division of the contents of the oöcyst into two masses, he did not distinguish the *Eimeria* from the *Isospora*, and grouped them all as *Psorospermium avium*. Rivolta and Silvestrini (1873) referred to this coccidium, and, as did Rivolta (1873), considered all the psorosperms of birds as belonging to one type. Rivolta (1878) gave the name *Gregarina avium intestinalis* to the parasite of fowls, and as this is an undoubted *Eimeria*, the specific name *avium* cannot be regarded as belonging to the *Isospora*, which was first definitely named *Diplospora lacazei* by Labbé (1893). It was referred to as *I. passerum* by Sjöbring (1897). Labbé (1899) gives a

list of about forty birds which have been recorded as infected with this coccidium.   Wasielewski (1904) examined 400 freshly caught birds in Germany, and found 20 per cent. infected.

Isospora lacazei (Labbé, 1893).—Though the oöcysts of this coccidium had been seen by Rivolta and Silvestrini, as shown above, and later by Rivolta and Delprato (1881) and Condorelli and Fiore (1892), Labbé (1893) was the first to give a clear account of its development, while Sjöbring (1897) and Laveran (1898a) gave a description of the main features of the life-cycle, which was again studied by Wasielewski (1904). It is possible that more than one species of *Isospora* occur in birds, as suggested by Labbé (1893).   At present there is no definite information upon which this point can be decided, as indeed Labbé (1896) recognized.
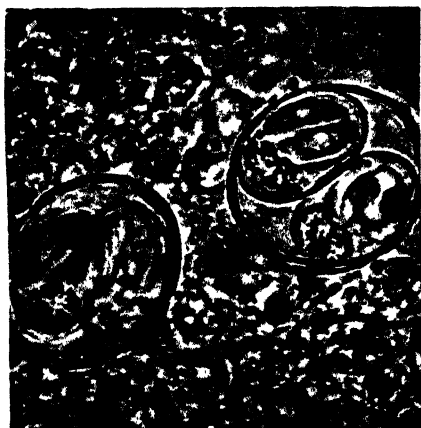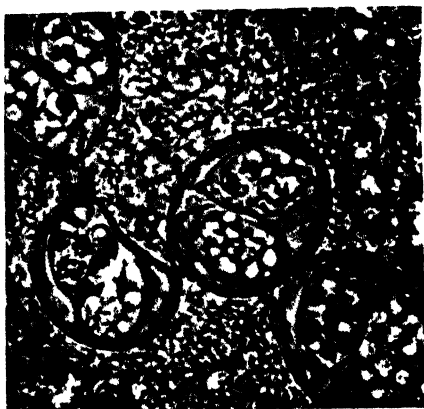
FIG. 351.—OÖCYSTS OF *Isospora lacazei* FROM THE INTESTINE OF BIRDS (× 1,000).   (AFTER WASIELEWSKI, 1904.)

Small birds, such as canaries, sometimes suffer from intense infections, which often prove fatal.   Within the intestinal epithelium the schizonts have a diameter of about 10 microns, and produce from 8 to 12 merozoites. These are 8 to 12 microns in length by 2 to 3 in breadth.   The microgametocytes, when fully grown, may measure 28 by 21 microns, and give rise to a large number of microgametes 2 to 4 microns in length.   As pointed out by Wasielewski (1904), it appears that the numerous microgamete nuclei result from repeated nuclear divisions during growth of the microgametocyte.   According to various observers, the oöcysts, which are ovoid bodies, vary in length from 16 to 30 microns (Fig. 351).   The two sporocysts in each oöcyst have a length of about two-thirds of that of the oöcyst, and are often more pointed at one end than the other.   At the

pointed end there is frequently seen a refractile globule. Within each sporocyst are produced four sporozoites and a large residual body. In its development *I. lacazei* resembles *I. felis* very closely.

ISOSPORA IN COLD-BLOODED VERTEBRATES.

The best-known member of the genus *Isospora* in a cold-blooded host is the form first described by Lieberkühn (1854) from the kidney of the frog (Figs. 353, 354). Labbé (1894) gave it the name *Klossia lieberkühni*, but two years later (1896) he changed it to *Hyaloklossia lieberkühni*. As pointed out above, the form seen by Eimer (1870) in the intestine of the frog, and which was named *Cytospermium ranæ* by Rivolta (1878), is not identifiable. Laveran and Mesnil (1902c) studied this parasite, and placed it in the genus *Isospora*. Other forms occur in reptiles. Hagenmüller (1898) described
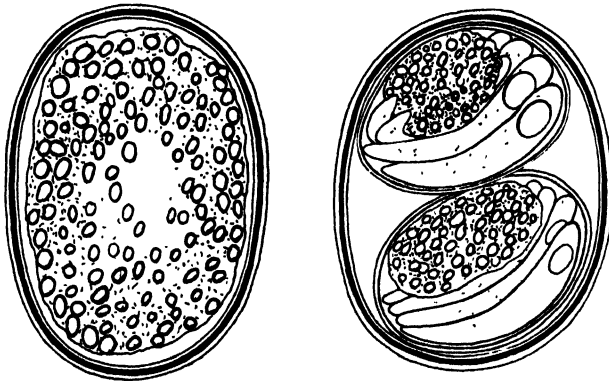
FIG. 352.—OÖCYST OF *Isospora* SP. FROM THE INTESTINE OF THE COMMON ENGLISH TOAD (×2,000). (ORIGINAL.)

I. camillerii from the lizard, *Gongylus ocellatus*, and *I. laverani* from the snake, *Cœlopeltis lacertina*, while Sergent (1902) gave the name *I. mesnili* to one which parasitized the nuclei of the intestinal epithelium of *Chamæleon vulgaris*. The oöcysts were about 30 microns in length, while the sporocysts measured 16 by 10 microns. Mesnil (1907) described as *I. hylæ* a coccidium of the rectum of the tree frog (*Hyla arborea*). The dimensions of the oöcysts were 30 to 35 by 20 to 25 microns. The writer found an *Isospora* in the liver and intestine of the lizard, *Agama colonorum*, in the Sudan. Another form with an oöcyst about 25 microns in length occurs in the intestine of the English toad (Fig. 352). Adler informs the writer that he has seen an *Isospora* in the lizard, *Varanus griseus*, in Palestine. It develops in the subepithelial tissues of the small intestine.

Isospora lieberkühni (Labbé, 1894).—This parasite, which occurs commonly in the kidney of the edible frog, *Rana esculenta*, has been studied by

FIG. 353.—STAGES IN THE DEVELOPMENT OF *Isospora lieberkühni* AS SEEN IN SECTION OF THE KIDNEY OF THE FROG. (AFTER NÖLLER, 1924.)

1. Groups of merozoites resulting from schizogony in the epithelial cells of the kidney tubules (× 950).   2. Macrogametocytes in the tubular epithelium (× 950).
3. Microgametocytes with numerous nuclei (× 950).
4. Microgametocyte with numerous microgametes (× 2,500).   5. Microgamete (× 2,500).

Laveran and Mesnil (1902c) and Nöller (1913a, 1923). According to Laveran and Mesnil, within the remarkably short period of forty-eight hours after ingesting oöcysts the frog shows an intense infection of the various organs of the body, with not only the asexually reproducing forms, but also the gametocytes. Nöller (1920b) believes that these frogs were probably suffering from an infection with *Lankesterella minima*, which reproduces in the endothelial cells of the capillaries and produces gametocytes in the same cells. According to Laveran and Mesnil, the oöcyst has a length of about 40 microns, and produces two ovoid or pointed sporocysts 25 to 30 microns in length, each containing a large residual body and four sporozoites 25 microns in length (Fig. 354). Both Laveran and Mesnil and Nöller (1913a) noted that the escape of the sporozoites from the sporocysts could be easily observed by treating them with an emulsion of the intestinal epithelium or pancreas of well-nourished frogs.

FIG. 354. — *Isospora lieberkühni* FROM THE KIDNEY OF THE EDIBLE FROG (*Rana esculenta*) (× 1,000). (AFTER LAVERAN AND MESNIL, 1902.)

Partially and fully developed oöcysts.

In his latest publication, Nöller (1923) has given some details of the life-history of the parasite. The oöcysts deposited in the water are swallowed by tadpoles. The sporozoites make their way to the glomeruli of the kidneys, in the epithelium of which schizogony takes place. The resulting merozoites then invade the tubule epithelium, which is found heavily infected with schizogony stages in young frogs towards the end of April or during May (Fig. 353). A few weeks later macrogametocytes and microgametocytes with a large number of biflagellate microgametes appear in the tubule cells, and eventually oöcysts are produced in this situation. The older frogs may again ingest oöcysts and become reinfected, but the heaviest infections are found in the young frogs, as tadpoles which live permanently in water have a better opportunity of ingesting oöcysts. Nöller again asserts that the development of *I. lieberkühni* is limited to the kidneys, and that the forms described by Laveran and Mesnil in other organs belonged to *Lankesterella minima* (Fig. 380).

(3) *Sub-Family :* EIMERIINÆ.

This sub-family includes the genus *Eimeria*, which was founded by Aimé Schneider (1875a) for the coccidium of the intestine of the mouse,

which had been named " *Gregarina* " *falciformis* by Eimer (1870). Its correct name is *E. falciformis* (Eimer, 1870), and it is the type species of the genus. Rivolta (1878) referred to the rabbit coccidium as *Psorospermium cuniculi*, while the name *Coccidium*, introduced by Leuckart (1879) for the rabbit coccidia, becomes a synonym of *Eimeria*. Aimé Schneider (1881) gave the name *Orthospora propria* to a coccidium of the intestine of newts. As this form is an undoubted *Eimeria*, the name *Orthospora* is another synonym.

The parasites belonging to this genus are characterized by the production of more or less spherical, elliptical, or ovoid oöcysts, which, when mature, contain four sporocysts, each of which contains two sporozoites. Another genus, *Crystallospora*, was established by Labbé (1896) for a coccidium of marine fish, *Motella tricirrata* and *M. maculata* (Fig. 372, 2). The sporocyst differs from those of members of the genus *Eimeria* in being shaped like a double pyramid with short spines at the various angles. The genus *Goussia* was also founded by Labbé (1896) to include forms like *E. schubergi*, which have more or less spherical oöcysts and sporocysts. The wall of the sporocyst is formed of two applied valves which open like the pods of a bean instead of being composed of a continuous membrane, as in the case of *E. stiedæ*. The members of the genus *Goussia* are mostly parasites of cold-blooded animals, and the oöcysts complete their development before leaving the host. Though the character of the sporocyst appears to be one of generic value, it seems doubtful if these forms can be separated from those of the genus *Eimeria* at present, as in many cases the sporocyst wall has not been investigated from this point of view. Another genus is *Paracoccidium* of Laveran and Mesnil (1902b). It was founded for a coccidium of the edible frog, which produced oöcysts and four sporocysts in the usual manner. After the oöcyst is mature, the sporocysts break open and liberate the eight sporozoites, which remain free within the oöcyst (Fig. 369, 8-10). This, again, is hardly a generic distinction, as the sporocysts may sometimes behave in this manner in an undoubted *Eimeria*. The single species, *Paracoccidium prevoti* Laveran and Mesnil, 1902, is best retained in the genus *Eimeria*. Another genus is *Jarrina*, established by Léger and Hesse (1922) for a small coccidium of birds. The oöcyst is peculiar in having a kind of neck, while the wall is traversed by fine canals which give the surface a punctate appearance (Fig. 369, 1-2).

The sub-family Eimeriinæ includes a large number of species of the genus *Eimeria*, one of which (*Eimeria schubergi*) has been considered above as a type of the coccidia. They are found as intestinal parasites of horses, cattle, pigs, sheep and goats, rats and mice, and rabbits; domestic birds, such as fowls, geese, ducks, pigeons, pheasants, grouse, and others; cold-blooded animals, such as the frog, newt, tortoise, sala-

mander, fish; and occasionally invertebrates (Arthropoda). Dobell (1919*a*), in his revision of the coccidia parasite in man, concluded that three species of *Eimeria* occur in man, two as intestinal forms and one in the liver. Since then he named another form discovered by Snijders (1921). Thomson and Robertson (1926) have, however, shown that the three intestinal forms are merely oöcysts of previously named coccidia of fish which were present coprozoically in the human intestine.

Medical literature contains numerous records of coccidiosis of the skin and other tissues of man and animals, but it is clear that the structures regarded as coccidia are not of this nature.

The majority of the species of *Eimeria* are known only in the oöcyst stage. As the oöcysts of different species often resemble one another fairly closely, for the recognition of specific differences reliance has to be placed on the minute characters of the oöcysts. In cold-blooded animals the oöcysts complete their development before leaving the host, whereas in the case of warm-blooded animals development takes place outside the body. In the latter case, the period required for the development varies with different species. The shape, dimensions, and colour of the oöcyst are characters to be attended to, as also the degree of development of the micropyle and the appearance of the oöcyst wall. The method of formation of sporoblasts has to be noted, and the presence or absence of a residual body when the four sporoblasts separate. The shape and size of the sporocysts and the character of the micropyle are features which vary with the species. It is usually assumed that the dimensions of the sporocyst are more constant for any one species than are those of the oöcyst, but not infrequently it will be found that the sporocysts vary in the same proportion as the oöcysts, and it cannot be assumed that, merely because the size of the sporocysts varies within unusually wide limits, more than one species is involved.

EIMERIA IN RABBITS AND HARES.

Owing to the fact that infection of the rabbit's bile ducts with coccidia leads to the formation of white nodules, this condition early attracted the attention of observers. Carswell (1838) published a coloured drawing of the condition in a rabbit's liver, which he regarded as tubercle. Hake (1839), however, was the first observer definitely to describe and figure the oöcysts, which he observed in the liver and duodenum. Though Hake is usually regarded as the first observer to see coccidia, Dobell (1922) has pointed out that Leeuwenhoek, in an unpublished letter dated 1674, gave some indication that he had seen the oöcysts in the bile of rabbits. Hake believed the oöcysts to be pus globules, and the condition in the liver one of carcinoma. Nasse (1843) again described the tubercle-like lesions in the rabbit's liver and figured the unsegmented oöcysts. Remak (1845) was the first to note the presence of oöcysts in the intestinal mucosa of rabbits. Handfield Jones (1846) again described the oöcysts which he found in the lesions in a rabbit's liver. He regarded them as altered liver cells. Rayer (1846) and Dujardin, to whom he

showed them, believed them to be immature eggs of a trematode. Kauffmann (1847) first noted that if the oöcysts were kept in water the contents segmented into four separate bodies. Küchenmeister (1852), Kölliker (1854), Lieberkühn (1855), Vulpian (1858), Neumann (1866), Leuckart (1863), and others referred to these enigmatical bodies without discovering their parasitic nature. Stieda (1865), however, observed the development of the oöcyst or psorosperm, as he termed it. He noted that the four spherical bodies within it became elongated, and that each became enclosed in a membrane. Within this membrane (sporocyst) there was formed a spherical granular body, at the side of which was a rod-like structure with swollen ends. The latter were evidently the heads of the two sporozoites, which, being closely applied to one another, were regarded as a single structure. Stieda concluded that the oöcysts were early developmental stages of some unknown animal parasite. In the same year Lindemann (1865) definitely regarded the organism as a parasite, and, looking upon it as a gregarine, gave it the name *Monocystis stiedæ*. Other observers, including Reincke (1866), Waldenburg (1867), Roloff (1868), Lang (1868), and Rivolta (1869–1878), made contributions to the subject. Leuckart (1879) gave a review of previous work, but did not observe any further development than that described by Stieda. He noted that the sporocyst had frequently a small knob at one end. He, however, stated it as his belief that the coccidium of the intestine of the rabbit, owing to its smaller size, a fact previously noted by Kölliker (1854), was a different species from that of the liver. He gave the name *Coccidium oviforme* to the one in the liver, and the name *C. perforans* to that in the intestine. As Lindemann (1865) had named the form in the liver *M. stiedæ*, and Aimé Schneider (1875a) had founded the genus *Eimeria* for the coccidium of the mouse, Leuckart's genus, *Coccidium*, is a synonym of *Eimeria*, and his two names, *C. oviforme* and *C. perforans*, are synonyms of *E. stiedæ* and *E. perforans*.

Balbiani (1884) investigated the development of the oöcyst, of which he gave an accurate description. He showed that the contents of the oöcyst segmented into four separate spherical bodies, and that each of these became elongate and encysted, as demonstrated previously by Stieda and Leuckart. He proved, however, that the sporocyst eventually produced a residual body and two sporozoites. Each sporozoite had a small central nucleus and a rounded end containing a refractile substance. The development of the oöcyst of *Eimeria stiedæ* was finally studied in detail by Metzner (1903).

As regards other stages of development, L. Pfeiffer (1890a, 1891) and R. Pfeiffer (1892) first pointed out that there occurred an endogenous multiplication, followed by the production of oöcysts within the epithelial cells of the rabbit's intestine, similar to that described by Kloss (1855) for *Klossia helicina* of the kidneys of snails, and by Eimer (1870) for *E. falciformis* of mice. He believed, as Eimer had done, that the oöcyst was responsible for the spread of infection from one animal to another, while the stages within the epithelial cells brought about an increase in the number of parasites in the host. These views were strongly contested by Schneider (1892), Labbé (1896), and others, who believed that the multiplying forms in the epithelium represented one parasite and the oöcysts another. Thus, so late as 1899 Labbé placed the schizogony stages of the mouse coccidium in one genus (*Eimeria*) and the oöcysts in another (*Coccidium*). A great advance was made by Schuberg (1895), who described almost completely the life-cycle of the coccidium of the mouse. He was the first to suggest that the minute bodies produced by certain stages of the parasite might be gametes destined to fertilize the large cells which developed into oöcysts. That these minute bodies were actually gametes was clearly demonstrated by Schaudinn and Siedlecki (1897) in the case of *Adelea ovata* and *E. lacazei*, two coccidia of the centipede.

Simond (1897) reared young rabbits from their birth on sterile milk. He fed them with ripe oöcysts, and demonstrated that in the resulting infection there occurred, not only oöcysts, but also the forms which were in reality schizonts, but which Schneider and others had insisted belonged to a distinct parasite. The facts regarding the life-history of these coccidia were finally placed on a firm foundation by the researches of Schaudinn (1900) on *E. schubergi*.

The life-cycle of coccidia having been elucidated, the complete development of the rabbit coccidium was described by Reich (1913), though Simond (1897), Wasielewski (1904), and Hadley (1911) had observed most of its stages. From these investigations it was found that the life-cycle of the rabbit coccidium very closely follows that of *E. schubergi*, as described by Schaudinn.

It has been shown above that Kölliker (1854) noted that oöcysts from the intestine of the rabbit were smaller than those from the liver, and that Leuckart (1879) had given the liver and intestinal forms distinctive names. The oöcysts of the intestinal form (*E. perforans*) measured about 24 by 12 microns, while those of the hepatic form (*E. stiedæ*) measured 32 to 37 by 15 microns. Leuckart noted that, though rabbits often showed infection of both the liver and intestine, some animals had a liver infection alone, and others the intestinal one only. Railliet and Lucet (1891a) furthermore noted that, during segmentation of the zygote into four sporoblasts, a residual body always occurred in the case of *E. perforans*, but never in *E. stiedæ*. R. Pfeiffer (1892), however, denied this difference, and noted that a residual body was sometimes present in both forms. Metzner (1903) and Reich (1913) maintained that a residual body was always present in the oöcysts of *E. stiedæ*. Metzner (1903) inoculated rabbits directly into the duodenum with oöcysts from the liver of other rabbits, and claimed that he always produced an intestinal infection. Lucet (1913), on the other hand, conducted a careful series of experiments with young animals free from previous infection. He fed them with oöcysts from the liver, and in all cases a liver infection alone was acquired, the animals dying in twenty-three to thirty-five days without any indication of intestinal infection. Lucet also noted that, apart from differences in size, the oöcyst of the intestinal form were colourless, while those from the liver had a yellow tint. According to Reichenow (1921a), there is not the least doubt that rabbits are liable to infection with two distinct species of *Eimeria*. The recognition of the two forms is not always an easy matter, for, though the majority of the oöcysts of *E. stiedæ* measure from 30 to 40 microns in length, occasionally smaller ones only 20 microns in length are met with. Furthermore, a residual body in the oöcysts of *E. stiedæ* does not appear to be a constant feature, nor are the oöcysts of *E. perforans*, which constantly produce a residual body, always colourless, as Lucet maintained. According to Reichenow (1921a), the shape of the oöcysts and the form of the micropyle are a more reliable means of distinction (Fig. 350, 1-3). The oöcysts of *E. stiedæ* are either ellipsoidal or ovoid in shape, while those of *E. perforans* are always ellipsoidal and often asymmetrical. Those of *E. stiedæ* are flattened at the micropyle end, while those of *E. perforans* are not. The micropyle is easily visible in the case of *E. stiedæ*, but it is difficult to detect in *E. perforans*. The oöcysts of *E. perforans* develop rapidly outside the body in about forty-eight hours, while those of *E. stiedæ*, under like conditions, take about seventy hours. Waworuntu (1924) has made a careful morphological and experimental investigation of the coccidia of the rabbit. He finds that the small form, *E. perforans*, is limited to the intestine, while the liver and intestine are liable to infection with three distinct types, one of which is *E. stiedæ*. The oöcysts of these three types are larger than those of *E. perforans*, and differ from one another in dimensions, shape, and the presence or absence of residual bodies in the oöcyst and sporocyst (Fig. 361).

The question of individuality of *E. stiedæ* and *E. perforans* appears to have been placed beyond doubt by Pérard (1924, 1924a), who has succeeded in obtaining pure strains of the two species. The former, fed to young specially reared rabbits, produces only liver lesions, which prove fatal to the young animals in twenty-eight to thirty days, while the latter gives rise only to an intestinal infection, which kills the rabbits in nine to fifteen days.

Eimeria stiedæ (Lindemann, 1865).—Young rabbits and hares are specially liable to infection, and they frequently die in large numbers from acute hepatitis, which is caused by the active multiplication of the coccidium in the biliary epithelium. When an outbreak occurs in epidemic form, there may be a high rate of mortality. If the animals survive the acute stage, as they not infrequently do, the infection becomes of a chronic type, and it is found that the infection still persists, but is limited to certain areas of the liver, where the bile ducts have been dilated to form white nodules, which may have a diameter of half an inch or more. On section these nodules, which are scattered through the healthy liver tissue, are found to have the structure of a biliary adenoma, in the epithelium of which all stages of development of the endogenous cycle of the coccidium are found (Fig. 355). In very old infections the coccidia appear to die in these liver tumours, which gradually shrink and become fibrotic or even calcareous. Infection is spread by oöcysts which escape into the intestine and are passed in the fæces, or by the dissemination of oöcysts after the death of the animal. It is possible that they may pass through the intestine of some carnivorous animal which has devoured the rabbit.

The development of *E. stiedæ* is most readily studied in sections of the liver (Fig. 355). In the cells lining the dilated ducts will be found every stage in growth from the newly entered merozoite up to the fully formed schizont which is breaking up into a very variable number of merozoites again. The merozoites escape into the lumen of the bile duct after rupture of the enclosing membrane, which is merely the remains of the host cell. They enter other cells, and the process of schizogony is repeated. Eventually, certain merozoites develop in the cells into micro- and macrogametocytes. The mature microgametocyte is the largest stage of the parasite, and consists of a mass of clear cytoplasm in which several large vacuoles occur. Large numbers of nuclei are produced, and these arrange themselves on the surface as clusters of fine chromatin granules. Each cluster becomes more compact, and finally assumes the characteristic comma shape. Finally, the microgametes, provided with two flagella which arise from the blunt anterior end, as first demonstrated by Wasielewski (1898), swim away into the lumen of the dilated bile duct in search of a macrogamete.
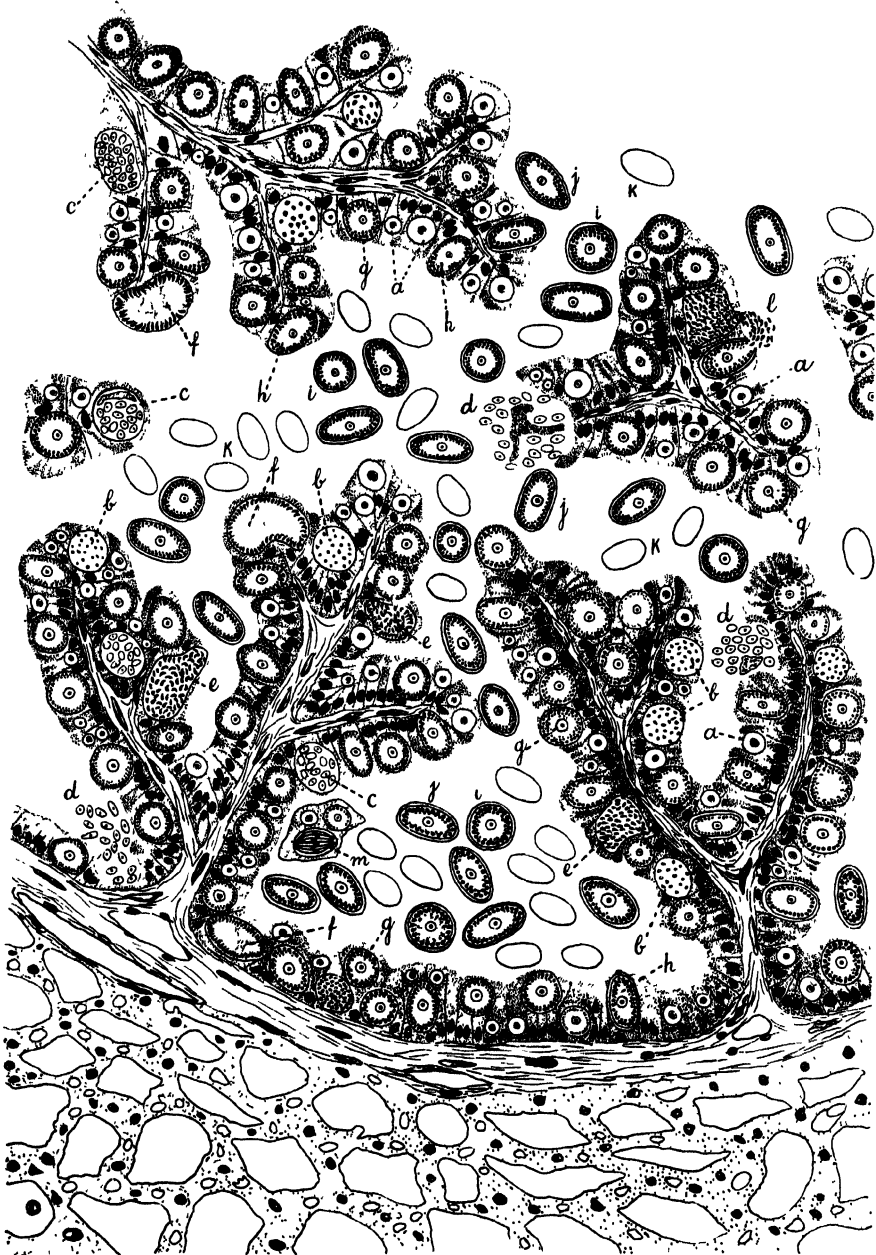
FIG. 355.—DIAGRAM OF APPEARANCE OF A SECTION OF PORTION OF A DILATED
    BILE DUCT OF THE RABBIT'S LIVER INFECTED WITH *Eimeria stiedæ*, SHOWING
    HYPERTROPHY OF EPITHELIUM AND VARIOUS STAGES OF DEVELOPMENT OF THE
    COCCIDIUM (× *ca.* 300).   (ORIGINAL.)

[*For description see opposite page.*

The macrogametocyte, when fully grown, is an ovoid body filled with globules of refractile material. The nucleus is central in position, and consists of a nuclear membrane, on which chromatin granules are arranged, and a central karyosome. The oöcyst is formed before fertilization, an opening or micropyle being left at one end. Through the latter the microgamete enters, its nucleus uniting with the macrogamete nucleus, which, as in *E. schubergi*, assumes the spindle form during fertilization. The oöcyst wall then becomes much thicker and shows a definite double contour. It is in this stage that the oöcysts are ready to leave the body (Fig. 356). If they do not escape from the liver, they remain there unchanged till they either degenerate or some chance circumstance brings about their release. The oöcysts which have escaped undergo the usual development on the ground (Fig. 357). The oval mass of granular cytoplasm filling the oöcyst contracts to a spherical body, which then becomes more or less square in outline, with the drawn-out corners consisting of clear cytoplasm, terminating in a granule which is eventually thrown off. This stage has been called the pyramid stage, as it may be regarded as made up of four pyramids united at their bases. The apex of each pyramid is composed of the hyaline cytoplasm. Four masses of cytoplasm, the pyramids, then separate. There may or may not be a residual body. The four sporoblasts thus formed then elongate, and measure from 14 to 18 microns in length by 6 to 8 microns in width. Round each of these is formed the sporocyst, which differs from that of *E. schubergi* in that it is a continuous sheath and is not composed of two convex valves. It is slightly more pointed at one end than the other. At the pointed end there is frequently seen a small knob, and this represents the micropyle, through which the sporozoites eventually escape. Each sporoblast divides into two sporozoites and a residual mass of granular cytoplasm. The sporozoites, which have centrally placed nuclei, are slightly curved structures with a pointed anterior end and a rounded posterior end in which a refractile body of homogeneous structure occurs. This stage being reached, the oöcysts are ready to infect other rabbits. According to Metzner (1903), the sporozoites escape from the sporocysts, make their way through the

a. Young forms which become either schizonts or gametocytes.
b. Full-grown multinucleate schizonts.
c. Merozoites still within cells after schizogony.
d. Liberation of merozoites after rupture of cell.
e. Mature microgametocyte, with numerous microgametes on its surface.
f. Section out of middle of mature gametocyte, with microgametes at periphery.
g. Transverse section of macrogametocyte within epithelial cell.
h. Longitudinal section of macrogametocyte within cell.
i. Transverse section of liberated oöcyst.
j. Longitudinal section of liberated oöcyst.
k. Oöcysts which are unstained owing to the fact that they were uncut.
l. Free microgametes, one of which is fertilizing a macrogamete.
m. Schizont with merozoites in " barrel " arrangement.

micropyle of the intact oöcyst under the influence of the pancreatic fluid, and enter the cells of the small intestine to commence the schizogony cycle. From the work of Pérard (1924a) and others it seems that the sporozoites do not invade the intestinal cells, but pass directly to the bile ducts. Reich (1913), who has studied the development of the rabbit coccidium, believes that two cycles of schizogony occur. One is the ordinary asexual multiplication terminating at each generation in about sixteen merozoites Eventually merozoites grow into another form of schizont, which breaks up into only four merozoites, which are remarkable in that they are flagel-

FIG. 356.—OÖCYSTS OF *Eimeria stiedæ* FROM THE CONTENTS OF A NODULE IN THE LIVER OF THE RABBIT (× 1,000). (AFTER WASIELEWSKI, 1904.)

lated and have a leptomonas-like structure. These enter other cells and develop into micro- or macro-gametocytes. It seems very doubtful if this description is correct, for, in the writer's experience, the number of merozoites produced varies from about six to thirty or more. Wasielewski (1904) stated that as many as a hundred merozoites are sometimes formed, but this may be a result of a cell containing several schizonts. The so-called flagellum described by Reich may be merely the appearance resulting from the adhesion of glutinous material to one extremity of the moving merozoite, a not uncommon phenomenon. The statement that the schizogony resulting in four merozoites is that which directly

precedes gametocyte formation requires confirmation. It is evident that, as in other coccidia, the number of merozoites produced varies considerably.

According to Waworuntu (1924), the merozoites are 8 to 10 microns in length. In the formation of the microgametes from the micro-gametocyte, Reich describes the central nucleus as giving off numerous small buds (heteropolar mitosis) into the cytoplasm. While this is
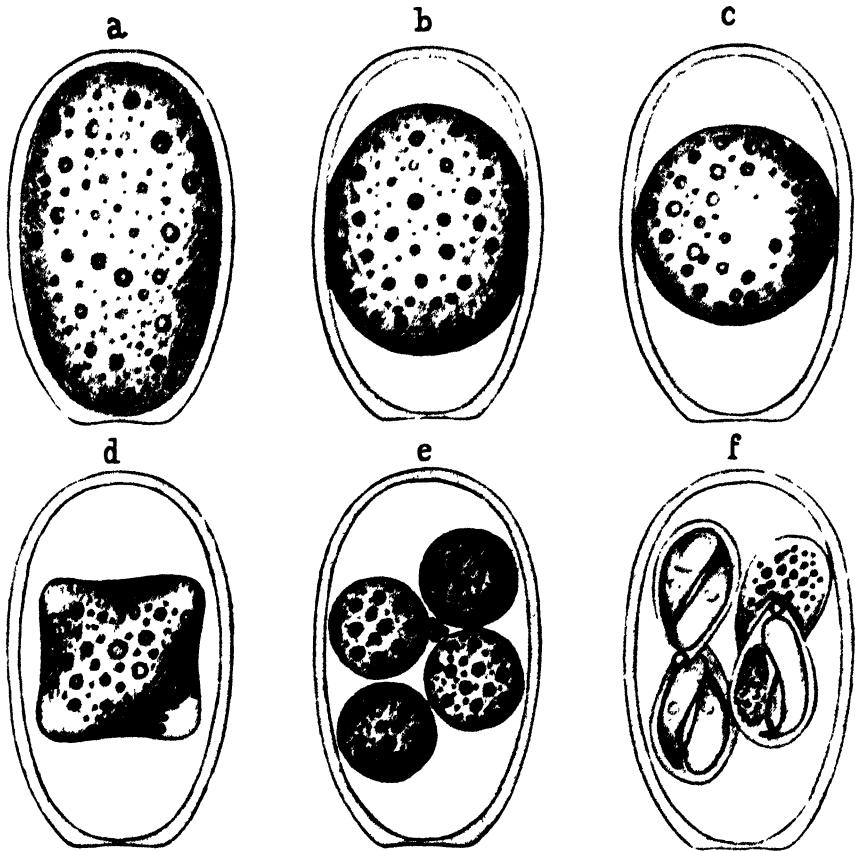
FIG. 357. —DEVELOPMENT OF THE OÖCYST OF *Eimeria stiedæ* OF THE RABBIT'S LIVER (× 1,250). (AFTER WASIELEWSKI. 1904.)

taking place, the whole nucleus breaks up into granules, which stream through the cytoplasm to collect in groups on the surface, while the degenerate remains of the nucleus maintain the original central position. It seems more probable, however, that, as in other coccidia, the micro-gamete nuclei arise by repeated mitotic divisions, the granules being chromosomes.

According to Reichenow (1921a), the oöcysts of *E. stiedæ* are 20 to 40 microns in length by 16 to 25 microns in breadth. Waworuntu (1924) gives 28 to 44 by 21 to 30 microns as the limits, and Pérard (1924a) 33 to 43 by 18 to 30 microns. According to the last-named observer, 90 per cent. of the oöcysts vary in length from 35 to 40 microns. They are ellipsoidal or ovoid in shape, distinctly flattened at one pole, and of a yellowish colour. There is usually a definite and easily detected micropyle at the flattened pole. It is represented by an opening in the thick inner membrane (Fig. 350, 1, 2). Outside the body the oöcyst becomes mature in about seventy hours. The sporocysts are ovoid bodies 14 to 18 microns in length. Each contains a definite residual body and has a knob-like thickening at one pole. When the sporoblasts form, according to Waworuntu, a residual body is also left in the oöcyst, but Pérard (1924a) points

1   *2*   *3*
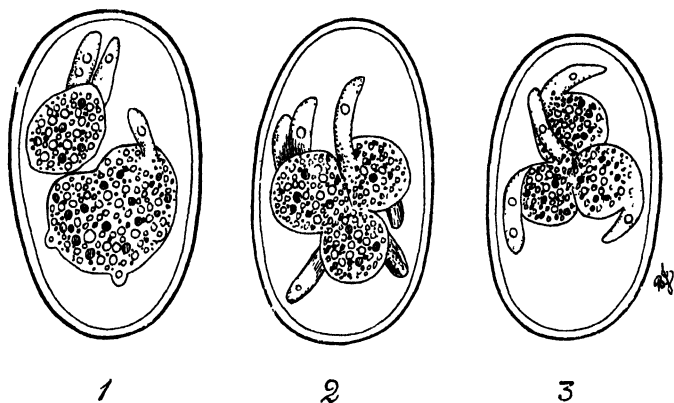
Fig. 358.—Oöcysts of *Eimeria stiedæ*, showing Abnormalities of Sporogony (× 1,300). (After Wasielewski, 1904.)

out that a definite spherical residual body like that occurring in *E. perforans* does not occur.

As is true of coccidia in general, the oöcysts are very resistant structures and highly impermeable. The writer fixed a smear of fæcal contents in sublimate solution, stained it with hæmotoxylin, and mounted it in balsam after dehydration and clearing. None of the reagents had entered the oöcysts, some of which completed their development up to the sporozoite stage on the slide.

Wasielewski (1904) pointed out that the oöcysts occasionally developed in an abnormal manner (Fig. 358). The contracted zygote or the four sporoblasts without the formation of sporocysts gave rise to sporozoites in an irregular manner.

Possible Occurrence in Man.—Oöcysts of a coccidium which has been identified with *E. stiedæ* have been recorded from the liver of man. According to Dobell (1919a), there are five authentic cases on record. The first is that of Gubler (1858). Leuckart (1863, 1879) gave a description of a case seen by Dresler, and reproduced Dresler's drawings of the oöcysts (Fig. 359). In 1879 he mentions two further cases, one of which was seen by Perls and Settler, and the other by Perls and von Sommering. The fifth case was described by Silcock (1890). From Dresler's drawings it appears that the oöcysts measured about 20 microns in length. They are thus smaller than the usual type of oöcyst found in the liver of rabbits. Silcock (1890) alone has seen the development of the human form into sporocysts, but he does not mention the number of these, nor the sporozoites. It is evident that the oöcyst bears some resemblance to the smaller forms of *E. stiedæ*, as Davaine (1860) noted, but the absolute proof of the identity of the human one with that of the rabbit is wanting. Guiart (1922) proposes to name this form *Coccidium gubleri* (=*Eimeria gubleri*).

Eimeria perforans (Leuckart, 1879).—This coccidium is limited to the intestine, as noted by Leuckart (1879), who was the first to recognize it as a different species from the form in the liver. Reichenow (1921a) gives the measurements of the oöcysts as 16 to 23 by 12 to 16 microns, while Waworuntu's figures are 15 to 30 by 11 to 18 microns.

FIG. 359. — Oöcysts of *Eimeria gubleri* FROM THE LIVER OF MAN. (*a*, × 330; *b* AND *c*, × 1,000). (AFTER LEUCKART, 1879.)

They are almost colourless and elliptical in shape, with a tendency to asymmetry (Fig. 350, 3). There is no flattening of the pole where the micropyle is situated, and the latter opening is very difficult to detect. The development outside the body is completed in forty-eight hours (Fig. 360). The sporocyst, which measures 10 to 12·5 microns in length, is an ovoid body with a knob at one end. There is a large residual body in the oöcyst and a small one in the sporocyst. Waworuntu (1924) has studied the development of *E. perforans* in the intestine, and finds that in all its stages it is smaller than those of *E. stiedæ*. from which it can be distinguished. The merozoites vary in length from 5 to 6·9 microns. Pérard (1924a) found that the vast majority of the oöcysts were between 24 and 30 microns in length, and that the average dimensions were 25·5 by 15·5 microns. Occasionally, in adult rabbits oöcysts as small as 10 by 8 microns were seen, but young rabbits infected with these revealed only oöcysts of

normal dimensions. Sometimes oöcysts as large as those of *E. stiedæ* were met with. Pérard thinks they may represent a variety of the usual type, and proposes the name *E. perforans* var. *magna*. They may represent the large intestinal forms referred to by Waworuntu (1924). Pérard noted that in *E. perforans* the fully developed oöcyst always contained a spherical residual body in addition to the four sporocysts. As regards the micropyle, it is only visible in certain oöcysts. When present, it is
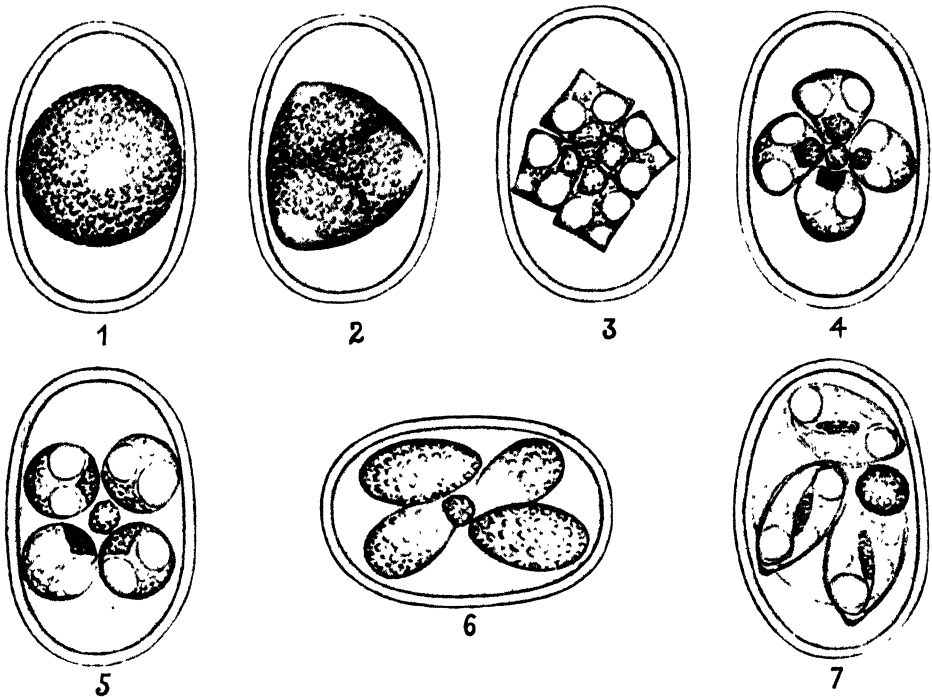
FIG. 360.—VARIOUS STAGES IN THE DEVELOPMENT OF THE OÖCYST OF *Eimeria perforans* (× 2,160). (AFTER WAWORUNTU, 1924.)

represented by an opening in the thick inner coat of the wall, the thin outer covering being continued over it.

Bruce (1919) in America has described what he regards as a distinct coccidium, which produces acute and fatal enteritis of young rabbits. It is apparently limited to the intestine, and produces oöcysts which vary in length from 15·77 to 39·84 microns and in breadth from 16·62 to 24·90 microns. The wall is thick, and in the larger oöcysts is of a pinkish orange colour. The oöcyst wall may be thickened by a deposit of material on it. This may cover the whole oöcyst or be limited to special areas. It

is especially evident around the very distinct micropyle.   Within the
oöcyst, when sporoblast formation takes place, a residual body, which
may be larger than the sporoblasts, is formed.   The sporocyst contains
a well-marked residual body also.   The nuclei of the sporozoites appear of
a pinkish colour.   Whether Bruce was dealing with a mixed infection of
E. stiedæ and *E. perforans*, or a race of one of these with an exceptionally
large range in the size of the oöcyst, or with a distinct species, cannot be
determined.

Waworuntu (1924) arrives at the conclusion that, in addition to
E. stiedæ and *E. perforans*, rabbits harbour two other forms (Fig. 361),
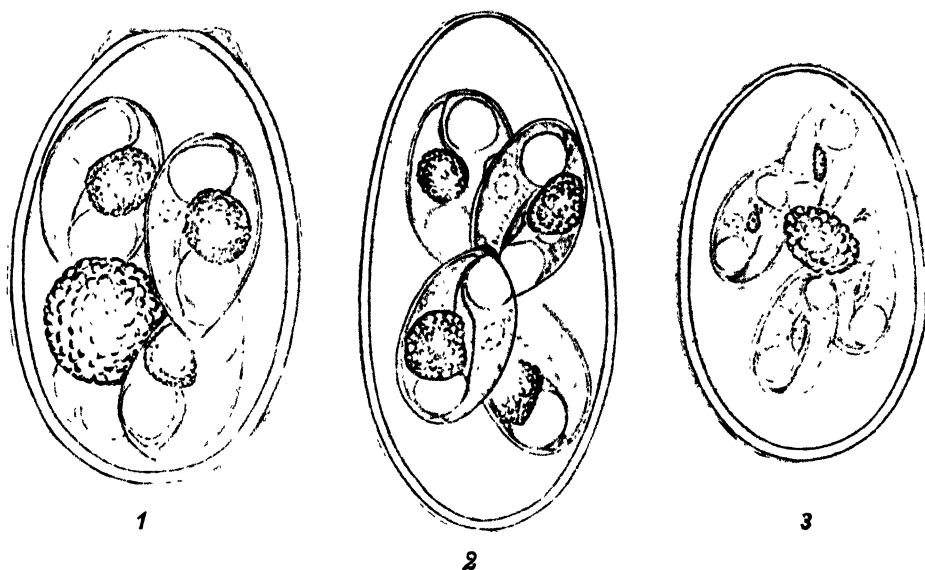
FIG. 361.— MATURE OÖCYSTS OF THE THREE TYPES OF COCCIDIA FROM THE LIVER
AND INTESTINE OF THE RABBIT (× ca. 1,600).   (AFTER WAWORUNTU, 1924.)

1. *Eimeria stiedæ.*   2. *Eimeria* sp.   3. *Eimeria* sp.

which can be distinguished by their dimensions and the residual bodies.
That these are actually distinct from *E. stiedæ* cannot be considered as
finally established.

A coccidium of the intestine and liver of hares was discovered by
Nieschulz (1923c) in Holland.   The oöcyst differs from that of *E. stiedæ*,
with which the hares were also infected, and measures 26 to 38 by 13 to 20
microns.   It was not possible to infect tame rabbits with this coccidium,
nor was it discovered in a large number of wild rabbits examined in the
same locality.   Nieschulz named the coccidium *Eimeria leporis*.

EIMERIA IN CATTLE.

An acute enteritis or dysentery of cattle associated with the presence of a species of *Eimeria* in the intestine occurs in various parts of the world. The parasite was first seen by Zürn (1878), and given the name *Cytospermium zürnii* by Rivolta (1878). The correct name for the parasite becomes *E. zürnii* Rivolta, 1878.

Eimeria zürnii (Rivolta, 1878).—Zschokke (1892), Hesse (1892), and Guillebeau (1893) were the first to describe a definite coccidiosis of cattle as a distinct disease in Switzerland. Guillebeau noted that in certain years the disease became epidemic, and led to considerable mortality amongst infected animals. Active multiplication of the coccidium occurred in both the large and small intestine, in the epithelium of which all stages of development could be found. The oöcysts described by him resembled those of *E. stiedæ* in dimensions, and in the presence of a distinct micropyle. Züblin (1908) studied the development of the oöcyst outside the body, and observed the production of four sporocysts, each with two sporozoites. He noted that the majority of oöcysts varied in diameter from 12 to 25 microns, while rarely larger forms—30 to 35 microns in length by 20 microns in breadth—occurred.

FIG. 362.—OöCYST OF *Eimeria zürnii* OF CATTLE (× 2,000). (MICROPHOTOGRAPH BY DR. LESLIE SHEATHER.)

Jowett (1911) studied the disease in South Africa, and gave as measurements of the oöcyst a length of 14·4 to 27·2 microns and a breadth of 12·8 to 20·8 microns. In Jowett's cases, the oöcysts resembled *E. perforans* of rabbits rather than *E. stiedæ*, but differed in that no residual body was formed when division of the zygote into sporoblasts took place. It will thus be seen that Guillebeau's dimensions are greater than those of Jowett, so that, as in the case of the rabbit coccidia, there may be two species represented. This is borne out by the observations of Theobald Smith and Graybill (1918) on coccidial dysentery of calves in America. According to these observers, the infection is contracted by the calves soon after birth, and the first symptoms of actual dysentery appear three to six weeks later. The disease runs a

course of six to eight weeks. Most usually the oöcysts are of the type described by Jowett. They measured 13·1 to 28·7 by 12·3 to 20·5 microns. The sporoblasts, when first formed, are spherical, and are developed without any residual body. They soon become ovoid and secrete a sporocyst, which has a thick cap at one pole. Within each are produced two sporozoites which completely fill the sporocyst, and there is no residual body. The sporocysts measure 9·9 to 11 microns in length by 5·3 to 5·7 microns in breadth. A second type of oöcyst was more rarely seen, and this corresponds with that seen by Guillebeau and the larger one of Züblin (Fig. 362). It has a thick wall, is often brownish in colour, and measures from 25·8 to 41·8 microns in length by 16·4 to 24·6 microns in breadth. As in the smaller form, no residual body is developed when division into sporoblasts takes place. The sporocysts are very much like those seen in the smaller oöcysts, but in this case there is formed a definite residual body along with the two sporozoites. Davis and Reich (1924) in California found the oöcysts of the coccidium of cattle varying in length from 24 to 34 microns, and in breadth from 17 to 21 microns.

EIMERIA IN SHEEP AND GOATS.

Coccidiosis of sheep was first recorded in America by Curtice (1892), and by Stiles and Hesse in the same year. It was noted by M'Fadyean (1896) in England, and by Mazzanti (1900) in Italy. Moussu and Marotel (1901) studied the disease in France, while Baldrey (1906) observed it in India and Mason (1916) in the Egyptian Sudan. Moussu and Marotel (1901) named the coccidium *Coccidium faurei*.

Eimeria faurei (Moussu and Marotel, 1901).—The oöcysts are usually ovoid, and measure 20 to 40 by 17 to 26 microns. Spherical oöcysts 18 microns in diameter may also occur. A coccidial infection of lambs has been studied by Lerche (1921) in Germany, who found the oöcysts to measure 27 to 31 by 15 to 23 microns. There is a definite micropyle closed by a cap. The sporocyst, which measures 13 by 6 microns, has a micropyle at the more pointed end. A residual body may or may not be left within the oöcyst when the four sporoblasts are formed. Such a body, however, occurs regularly within the sporocysts. Davis and Reich (1924) in California noted that the oöcysts were ellipsoidal or ovoid in shape, measured 24 to 40 by 17 to 25 microns, possessed a bulging cap or nodule at the micropyle end and had no residual body. Spiegl (1925) has given the name *Eimeria intricata* to a form with larger oöcysts which have rough surfaces and conspicuous pole caps. Sheather (1926) saw the oöcysts of this species in lambs. They measured 42 to 60 by 31 to 44 microns, while the oöcysts of *E. faurei*, also present, measured 20 to 40 by 17 to 23 microns.

Coccidiosis of goats was first noted by Marotel (1905) in France. He

gave the name *E. arloingi* to the coccidium. It was again seen by Martin (1907) and Spiegl (1919) in Germany, by Stevenson (1911) in the Sudan, and by Velu (1919) in Morocco. According to Spiegl, the oöcysts measured 21 to 33 by 16·5 to 22·5 microns. It seems not improbable that *E. arloingi* is identical with *E. faurei* of sheep. In fact, Nöller, Schürjohann and Vorbrodt (1922) have conducted cross-infection experiments with sheep and goats, and claim to have established the identity of the two forms (Fig. 363).

Various symptoms, chiefly of an enteritic or dysenteric character, have been ascribed to coccidiosis. Healthy sheep and goats are commonly
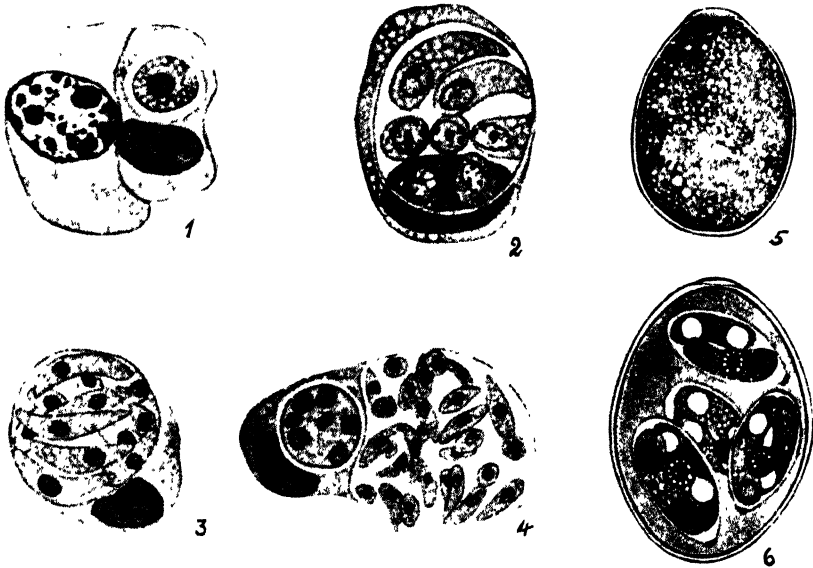
FIG. 363.—*Eimeria faurei* FROM THE INTESTINE OF THE GOAT. (AFTER NÖLLER, SCHÜRJOHANN, AND VORBRODT, 1922.)

1-4. Young schizont in epithelial cell and three types of merozoite (×2,400).
5-6. Immature and mature oöcysts (× 1,200).

infected, and when symptoms occur in association with large infections it is difficult, as in all such cases, to decide if the condition is primarily one of coccidiosis or not.

EIMERIA IN PIGS.

Pigs commonly harbour a coccidium of the genus *Eimeria*. Cauchemez (1921a), who suggested the name *E. brumpti*, points out that Johne (1882) saw this organism and regarded it as *E. stiedæ*, while Rivolta considered the form seen in the pig as *E. zürnii*. The coccidium referred to as *Coccidium jalinum* by Perroncito (1901) is merely *Blastocystis*. The first accurate description of the pig coccidium was that of Douwes (1921). He

proposed the name *E. debliecki*, while nine days later [according to Nieschulz (1922)], Krediet used the name *E. jalinum* for the pig coccidium. In the same year (1921) Nöller proposed the name *E. suis* and Cauchemez *E. brumpti*. The correct name is evidently *E. debliecki*, as Cauchemez (1922) admits.

Eimeria debliecki Douwes, 1921.—Douwes observed that the oöcysts of this coccidium were of two types. Large forms measured 50 by 35 microns, while smaller ones were 18 to 24 by 15 to 20 microns. He believed it possible that two distinct species were represented. Cauchemez states that the oöcysts are exceedingly variable in size, and gives 13 to
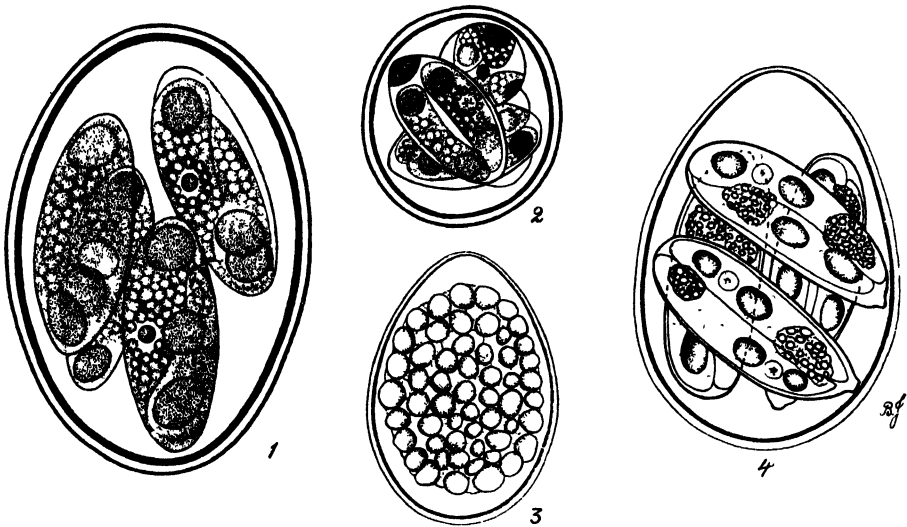
FIG. 364.--OÖCYSTS OF *Eimeria debliecki* OF THE PIG. (1 AND 2, AFTER NÖLLER AND FRANZ, 1922; 3 AND 4, ORIGINAL.)

1-2. Two extremes in size of mature oöcysts of German pigs (× 1,900).
3-4. Immature and mature oöcysts from English pigs (× 1,600).

30 microns as the length and 9 to 18 microns as the breadth. The oöcysts, which complete their development in seven to nine days, bear a close resemblance to those of *E. zürnii* of cattle (Fig. 364).

Nöller and Frenz (1922) studied *E. debliecki*, and gave as the measurements of the oöcysts 12 to 33 microns for their length and 10 to 20 microns as their breadth, while, judging from their figures, the sporocysts are also very variable in size. Those in oöcysts 31 microns in length are about 19 microns long, while those in oöcysts 15 microns in length are about 11 microns long. Nöller and Franz noted that oöcysts of all intermediate sizes occur, so that it is impossible to decide whether Douwe's view that

two species occur is correct or not. Davis and Reich (1924) gave measurements of 19 to 26 by 16 to 23 microns for the oöcysts from pigs in California.

EIMERIA IN CATS AND DOGS.

There are very few records of the occurrence of coccidia of this genus in carnivores, which appear to be much more commonly infected with species of *Isospora*. Virchow (1865) recorded the occurrence of psorosperms in the bile of a dog, but it is impossible to form an opinion as to the nature of these bodies. They may have been eggs of a trematode. Perroncito (1876) also described psorosperms from the bile ducts of a dog. They were named *Cytospermium hepatis canis familiaris* by Rivolta (1878), but they were clearly eggs of a trematode. Guillebeau (1916) described coccidia measuring 7 by 12 microns from the liver of a dog. In spite of their much smaller size, he identified them with *E. stiedæ* of the rabbit. It is not clear that they actually belonged to the genus *Eimeria*. Chierici (1908), however, recorded what appear to be undoubted *Eimeria* from the bile of a cat. The oöcysts, which were ovoid, varied in length from 26 to 30 microns and in breadth from 17 to 20 microns. Development resulted in the formation of four sporocysts, each with two sporozoites. Brown and Stammers (1922) recorded the presence of an *Eimeria* in the fæces of three dogs in London. It was described by the writer (1923a), who gave it the name *E. canis*. Another form, which occurred in a cat in Holland, and which may be identical with the one seen by Chierichi, was named *E. felina* by Nieschulz (1924a).

Eimeria canis Wenyon, 1923.—This coccidium, which was seen only in the oöcyst stage, was discovered in the fæces of three dogs in London (Fig. 365). Nieschulz (1924) has met with it in a dog in Holland. The oöcyst was ellipsoidal or ovoid in shape, and varied in length from 18 to 45 microns and in breadth from 11 to 28 microns. There was a thick wall, in many cases covered by an outer rough membrane of considerable thickness, which tended to separate from the true wall of the oöcyst. It was often represented by fragments adherent to the true oöcyst wall. In many cases no such outer membrane occurred. When present, it left one end of the oöcyst uncovered where a definite micropyle could be detected. A curious feature of the oöcysts was their red or pink colour, a feature which was noticed by Bruce (1919) in a very similar coccidium seen by him in rabbits in America (see p. 840).

The oöcysts completed their development in three or four days. The cytoplasm contracted to a sphere, which sometimes remained attached to the micropyle region by a pedicle. Lines radiating from the micropyle in some oöcysts appeared to indicate the presence of a fine inner membrane

lining the oöcyst. The division into sporoblasts was effected by the cytoplasmic mass becoming quadrangular. The angles of this mass then formed four pyramids with clear tips, which separated as sporoblasts, with or without the formation of a small residual body. The sporoblasts then became elongated and produced sporocysts, which frequently had a knob at one end. This knob undoubtedly represents the micropyle. Within the sporocysts, which vary in size with the oöcyst, are formed two sporozoites and a residual body.

In many respects *E. canis* resembles a mixed infection of *E. stiedæ* and *E. perforans* of the rabbit. In the range in size of the oöcysts it is
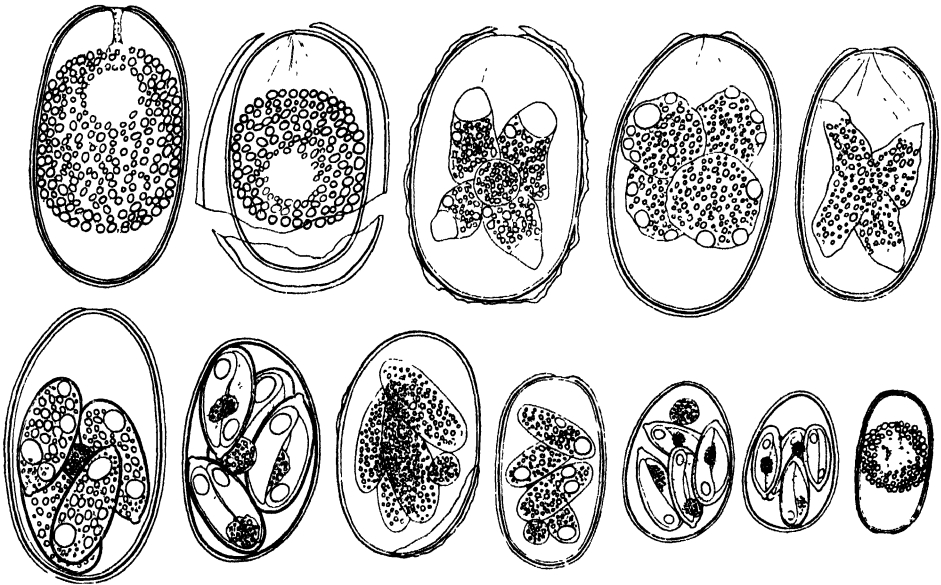
FIG. 365. —*Eimeria canis:* EXTRACORPOREAL DEVELOPMENT OF OÖCYSTS WHICH VARY CONSIDERABLY IN SIZE (× 970). (AFTER WENYON, 1923.)

similar to *E. debliecki* of the pig, as described by Cauchemez (1921*a*). The only coccidium which resembles it to any extent is the form described by Bruce from the rabbit. In this case the oöcysts were of a pinkish colour, and often had an outer covering in the form of a thick coarse membrane.

Eimeria felina Nieschulz, 1924.—This form was discovered by Nieschulz (1924*a*) in the fæces of a cat in Holland. The oöcysts, which measure 21 to 26 by 13 to 17 microns, resemble the intermediate-sized oöcysts of *E. canis*. They were, however, colourless, and did not have the thick outer membrane seen in many of the oöcysts of *E. canis*. Moreover, the variation in the size of the oöcysts was not so great as in those of *E. canis*.

On account of these differences, Nieschulz established the new species. Chatton and Blanc (1917) mention the occurrence of an *Eimeria* in the cat in Tunis. Merozoites were figured, but no other details were given.

EIMERIA IN RATS, MICE, AND OTHER SMALL MAMMALS.

The coccidium of the mouse was first seen and studied by Eimer (1870), who called it " *Gregarina* " *falciformis*. He described the process of schizogony, and conjectured that the oöcysts were destined to convey infection to new hosts. He mentions the occurrence of similar coccidia in rats. Though Eimer had expressed it as his opinion that the cysts containing a number of falciform bodies (merozoites) were merely stages in the development of the same parasite, which gave rise to the psorosperms (oöcysts), later writers did not accept this view, and thought that two distinct parasites were represented. Aimé Schneider (1875a) founded the genus *Eimeria* for the parasite in its schizogony stage, and named it *E. falciformis*, while Rivolta (1878) called it *Gregarina muris*. This parasite was referred to as *E. falciformis* by Pfeiffer (1890) and by Labbé (1896). It was studied by Schuberg (1892), who placed it in Leuckart's genus *Coccidium* as *Coccidium falciforme*. Jackson Clarke (1895) described certain stages in the intestinal epithelium of mice without understanding their significance. He, however, noted that the oöcyst developed four sporocysts, each with two sporozoites. Schuberg (1895) gave an excellent account of the development of the mouse coccidium. He described the process of schizogony, the formation of the oöcyst from the macrogamete, and the formation of numerous minute bodies by the microgametocyte, which he suggested might be destined to fertilize the macrogametes. Labbé (1899) took a retrograde step in describing as *E. falciformis* the schizogony stages of the parasite and as *C. falciforme* the oöcyst, though he suggested the possibility of the two being merely different developmental forms of one parasite. That this was actually the case was clearly recognized after Schaudinn (1900) had demonstrated the complete life-cycle of *E. schubergi*. Reich (1913) described stages in the development of the microgametes, the biflagellate nature of the microgamete, the development of the macrogamete, and its fertilization by the microgamete. The coccidium first mentioned by Eimer (1870) and later by Grassi (1881a) as occurring in rats has been regarded as identical with that of mice, but Nöller (1920b) failed to infect rats with ripe oöcysts obtained from mice, while Dieben (1924) appears to have proved it to be a distinct species. Labbé (1899) mentions the occurrence of a coccidium, which he regarded as a variety of *E. perforans*, in the guinea-pig, the hamster, *Cricetus frumentarius*, and *Mustela vulgaris*. Galli-Valerio (1905a) gave the name *E. arvicolæ* to a form in *Arvicola nivalis*. Later he (1922) named a form in the squirrel (*Sciurus vulgaris* var. *alpina*) *E. sciurorum*, and in the following year (1923) one in the marmot (*Arctomys marmota*) *E. marmotæ*. Nöller (1920b) named the coccidium of the hamster *E. falciformis* var. *criceti*. Lavier (1924a) has described as *E. hessei* a coccidium found in the intestine of the bat, *Rhinolophus hipposideros*.

Eimeria falciformis (Eimer, 1870).—This coccidium is a common parasite of mice. It develops chiefly in the epithelial cells of the small intestine, but may also occur in the large intestine and also the stomach, as pointed out by Jackson Clarke (1895). In severe infections Reich (1913) noted that the parasite invaded the subepithelial tissues. It gives rise to an acute enteritis. In experimental mice fed with ripe oöcysts on the fourth

and fifth day, Nöller (1920*b*) noted that the intestinal epithelium contained enormous numbers of schizonts. Oöcyst formation began about the fifth day. It was shown that, as the infection abated, a superimposed infection could be induced by again feeding the animals with ripe oöcysts. It was found impossible to infect rats or dogs with the coccidium of the mouse.

Mice in England are commonly infected with *E. falciformis*, and in the early stages of infection all phases of development are seen in sections of the intestine (Fig. 366). In later stages the only forms which can be found are the gametocytes, and in such cases it is possible to trace the
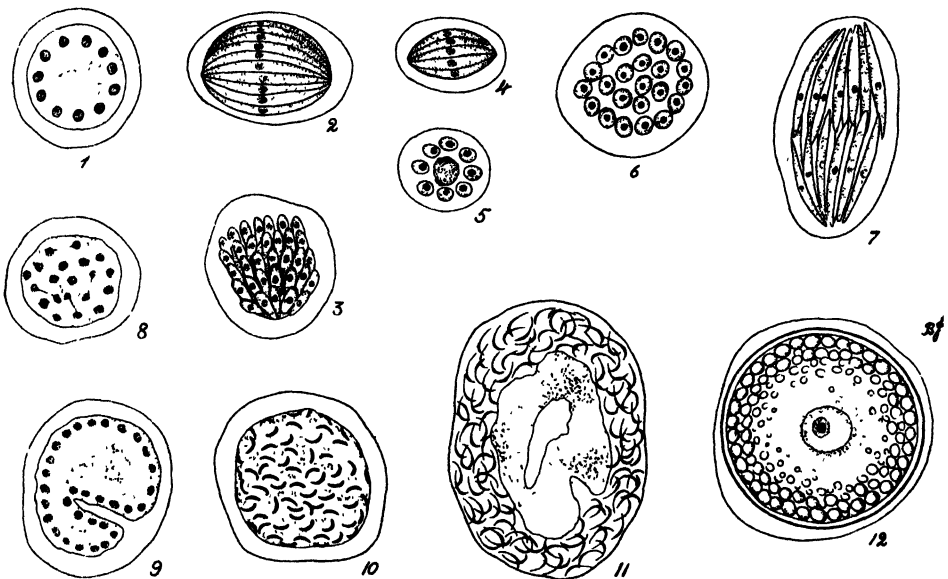
FIG. 366.—STAGES IN DEVELOPMENT OF *Eimeria falciformis* AS SEEN IN SECTIONS OF THE INTESTINE OF MICE (× 2,400). (ORIGINAL.)

1. Section of schizont.   2-4. Bundles of merozoites resulting from schizogony.
5-6. Section through bundles of merozoites.
7. Longitudinal section of bundle of merozoites.
8-11. Development of microgametocyte and formation of macrogametes.
12. Section of macrogametocyte.

development of the male gametocyte without fear of confusion with the developing schizonts.

The merozoites produced by the mature schizonts vary both in size and number. There may be only eight, and possibly less, or as many as thirty or more. They are elongate falciform bodies, which often lie round a central residual mass of cytoplasm like ribs of a barrel or segments of an orange. The number of merozoites produced varies with the size of the mature schizont. The male gametocyte, when mature, is a large irregularly shaped cell, on the surface of which are numerous nuclei.

These have arisen during the growth of the gametocyte by repeated nuclear divisions. Finally, there are formed numbers of comma-shaped microgametes each of which, according to Reich (1913), is provided with two flagella like the microgametes of *E. schubergi*. The microgametes, when first formed, lie in the vacuole in the host cell around a large residual body. The macrogametocyte is a large ovoid or almost spherical body containing a central nucleus and globules of refringent material in its superficial cytoplasm. Fertilization, which was described by Reich (1913), takes place after formation of the oöcyst around the macrogamete. The oöcysts are often almost spherical, and measure from 16 to 21 microns in length by 11 to 17 microns in breadth (Fig. 367). The
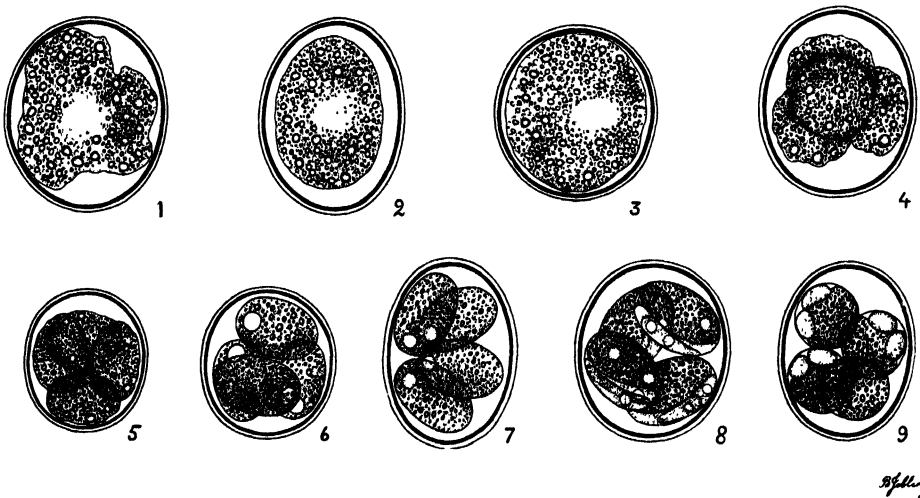
FIG. 367.—*Eimeria falciformis:* OÖCYSTS IN DIFFERENT STAGES OF DEVELOPMENT, SHOWING VARIATIONS IN SHAPE, FROM WHITE MICE (× 1,100). (ORIGINAL.)

development of the oöcyst outside the body is completed in five or six days. During the formation of the four sporoblasts a pyramid stage occurs, with a refringent granule at the apex of each pyramid, as in *E. stiedæ*. A small residual body may or may not occur in the oöcyst. The sporocysts have a breadth of about two-thirds of their length. There is a well-marked micropyle at one end of the sporocyst, within which are the two sporozoites and a definite residual body.

Eimeria nieschulzi Dieben, 1924.—It has usually been supposed that the *Eimeria* of the rat is identical with that of the mouse, but Dieben (1924) has shown that the oöcysts of the rat form measure 18 to 26 by 14 to 20 microns. They are thus larger than those of *E. falciformis*, and, furthermore, they differ in shape in that one end tends to be more pointed than the other. The merozoites vary in length from 7·5 to 26 microns, and in

breadth from 1·5 to 1·8 microns. Attempts to infect mice, guinea-pigs, and rabbits with the rat form failed, though it was possible to infect both *Rattus rattus* and *R. norwegicus*. On account of these observations Dieben, who has given a detailed account of its life-history, gave the name *E. nieschulzi* to the rat coccidium.

Eimeria caviæ Sheather, 1924.—The coccidium of the guinea-pig, which was first noted by Labbé (1899), who regarded it as *E. perforans*, is of fairly common occurrence. Strada and Traina (1900) stated that it occurred in the liver as well as in the intestine. Bugge and Heinke (1921) described the oöcyst as measuring 15·9 to 24·6 by 12·2 to 17·4 microns. Dieben (1924) stated that the oöcysts could be distinguished from those of the rat coccidium, and that it was not improbable that it is a distinct species. Sheather (1924) has described the guinea-pig parasite and given it the name *E. caviæ*. He found that the various stages resembled those of the coccidia of the rabbit, but occurred only in the large intestine.

EIMERIA IN MAN.

Though species of *Eimeria* are such common parasites of the intestine of domestic animals, the human intestine appears to be free from them. Dobell (1919a), in his paper already referred to, has reviewed the literature on the subject of human coccidiosis. Many of the structures recorded as coccidia from the skin and organs of man are not of this nature, and others have been so imperfectly described that their identity cannot be determined. Dobell (1919a, 1921a) concluded that apart from the genus *Isospora* there were four species of *Eimeria* parasitic in man. One of these (*E. gubleri*) has been mentioned above (p. 839). The others, the oöcysts of which were found in human fæces, were named *E. wenyoni*, *E. oryspora* and *E. snijdersi*. It now transpires from the work of Thomson and Robertson (1926) that, as the writer had long suspected, these are not human parasites, but merely *E. clupearum* and *E. sardinæ* of herrings, the oöcysts of which had been ingested and were passing through the human intestine. The possibility of this was stated by the writer in the original manuscript of this treatise, and it explains the fact well expressed by Dobell (1921a) in the following words: " Their cysts have suddenly appeared in the stools and then promptly vanished—never to return."

Eimeria clupearum (Théolhan, 1892).—This coccidium, which was named *E. wenyoni* by Dobell (1919a), who considered it to be a human parasite, was discovered by the writer (1915g) in the fæces of a patient returned to England from Gallipoli. The oöcysts were not present in large numbers, but one or two could be found in each cover-glass preparation. What was possibly the same coccidium was later seen by Roche

(1917) in three cases in Salonika, while Knowles (1924) records a case from Calcutta. The oöcyst is spherical, and has a diameter of about 20 microns (Fig. 350, 11). It possesses a fairly thick wall, which is roughened on its outer surface and lined by a more delicate membrane on its inner surface. It is of a light brown or yellow colour. Four sporocysts occur, each having rounded ends and measuring about 10 by 7 microns. They are roughened on their outer surface. Each sporocyst contains two sporozoites and a residual substance in the form of one or more masses of refractile material. Each sporozoite, with rounded anterior and pointed posterior end, contains an ovoid refractile body in its anterior portion.

The writer had already suggested in the manuscript of this work that there was a possibility that Dobell's *E. wenyoni* was merely Théolhan's *E. clupearum* (see p. 860). Thomson and Robertson (1926) have now shown that the oöcysts of *E. clupearum* occur in large numbers in the livers of herrings, sprats and mackerels. There is no question that this is the source of the oöcysts in the human fæces. Dobell's name *Eimeria wenyoni* becomes a synonym of *E. clupearum* which was described and figured by Thélohan (1892, 1894).

Eimeria sardinæ (Thélohan, 1890).—This coccidium, which was seen by Dobell on two occasions in the fæces of a patient who had contracted amœbic dysentery, was, on the assumption that it was a new human parasite, named by him *Eimeria oxyspora*. It was present in extremely small numbers. Only the oöcysts were seen, and these were passed in the fæces in the fully developed form. The oöcyst was spherical, with a diameter of about 36 microns, and possessed of a wall composed of two distinct layers—an inner fairly thick and uniform, and an outer which appeared composite, and encrusted with adherent bacteria and other foreign particles from the fæces. The wall as a whole was faintly yellow in colour, but quite transparent. The four sporocysts were sharply pointed at each end, and measured from 30 to 32 microns in length by 7·5 microns in breadth. The sporocyst was composed, as usual, of a tough inner coat (endospore) and a deciduous thin outer one (epispore). The two sporozoites within each sporocyst were long and slender, with rounded posterior and pointed anterior ends. The rounded end of each sporozoite contained an ovoid body. Between the latter, which Dobell considered to be the nucleus, and the blunt posterior end of the sporozoite were two or three bright fusiform bodies. A few granules occurred in the cytoplasm anterior to the nucleus. The residual body was represented by a few scattered granules lying near the middle of the sporocyst. Similarly, the residual body of the oöcyst was represented by a small number of granules lying outside the sporocysts.

A similar case in a woman who had never been out of Europe save for

two months spent in Malta in 1920 was studied by Broughton-Alcock and Thomson, J. G. (1922). This case, the second to be described, differed from that studied by Dobell in that large numbers of oöcysts were present in the fæces in all stages of development (Fig. 368). These varied in
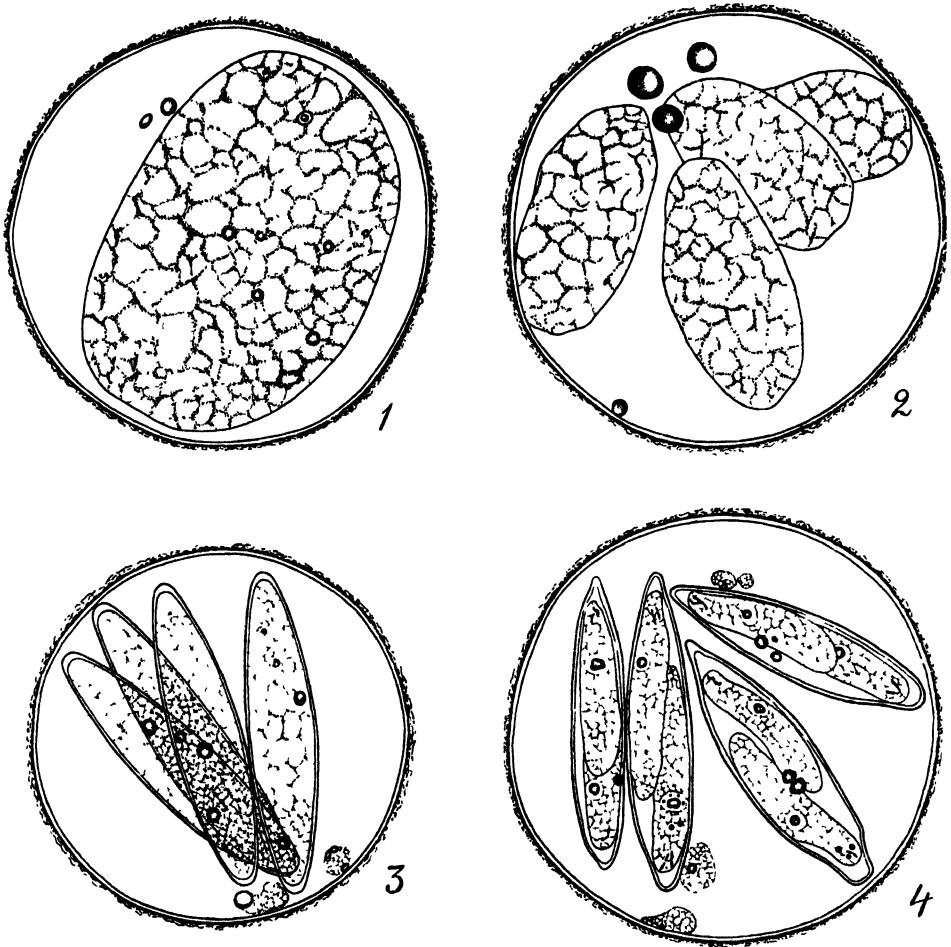
FIG. 368.—*Eimeria oxyspora* (= *E. sardinæ*): OÖCYSTS FROM BROUGHTON-ALCOCK AND THOMSON'S CASE (× 1,300). (ORIGINAL.)

1. Oöcyst with somewhat retracted zygote.
2. Oöcyst with four sporoblasts and globules representing a residual body.
3. Oöcyst with four sporocysts and residual bodies.
4. Mature oöcyst with four sporocysts, each with two sporozoites and globules representing residual bodies.

diameter from 33·6 to 50·6 microns, with an average of 42·5 microns. The zygote segmented into four pyramidal sporoblasts and some residual cytoplasm. The sporoblasts became elongated and secreted sporocysts, which varied in length from 25 to 30 microns and in breadth from 6 to 7 microns.

The sporoblasts divided into two sporozoites and some residual cytoplasm. The length of the sporozoites was, on an average, 19·6 microns and the breadth 3 microns. Slightly in front of the centre of the sporozoite a small spherical nucleus with a central karyosome could be detected. Some of the sporozoites showed the short fusiform structures which Dobell described as occurring at their rounded anterior ends, while some, again, possessed an ovoid refractile body in this region. The latter was regarded by Dobell as the nucleus, which, however, is more central in position and less clearly visible. The ovoid refractile body is very frequently present in the anterior part of the sporozoites of coccidia. A third case has been recorded by Thomson and Robertson (1922) in England.

As in the case of *E. clupearum* (*E. wenyoni*) the work of Thomson and Robertson (1926) has shown that this organism is not a human parasite, and that the oöcysts are merely those of *E. sardinæ* described by Thélohan (1890) as occurring in the testis of the sardine. Thomson and Robertson have found it in large numbers in the " soft roe " or testis (both fresh and tinned) of the herring and sprat, which are common articles of diet. The oöcysts, as suggested by the writer in his manuscript, were merely passing through the human intestine.

In this connection it is necessary to mention another coccidium, only the oöcysts of which have been found in human fæces (Fig. 350, 12). It was discovered by Snijders (1921) in Sumatra in a case of amœbic dysentery, but was seen only on one occasion. It has been described by him (1921) and also by Dobell (1921a), who examined some of Snijder's material and gave the organism its name.

The oöcysts occurred uniformly in all parts of the fæces, and were spherical transparent bodies measuring 40 to 48 microns in diameter. There was an inner wall surrounded by an ill-defined mucous layer. Four pointed sporocysts occurred in each oöcyst, and they measured from 20 to 25 microns in length by 7 to 8 microns in breadth. The sporozoites were elongate bodies pointed at one end (anterior) and blunt at the other (posterior). The residual body of the oöcyst was represented by a few vestiges, but that of the sporocyst was more definitely present in the form of one or two small, highly refractile, spherical bodies of varying size. The majority of the oöcysts were fully developed in the stool, which had been passed only half an hour before examination. A certain number of incompletely developed oöcysts were, however, also present. It was found that many of the oöcysts were in a degenerate condition.

It will be noted that this coccidium was regarded by Dobell as a distinct species, chiefly on account of the measurements of the oöcysts and sporocysts, which differed from those of the form he named *E. oxyspora*. The case studied by Broughton-Alcock and Thomson enabled them to

demonstrate that there was a much greater variation in the size of the oöcysts and sporocysts than Dobell had noted in the case studied by him. Furthermore, the material on which the measurements of *E. snijdersi* were made had been fixed and mounted, a procedure which is not calculated to give accurate pictures of oöcysts. The size of the oöcysts of *E. snijdersi*, as given by Dobell, fall within the range of those of *E. oxyspora*, while the largest sporocysts of *E. snijdersi* are equal to the smallest of *E. oxyspora*. It seems, therefore, not improbable, as suggested by Broughton-Alcock and Thomson, that *E. snijdersi* and *E. oxyspora* are identical, and that the differences noted by Dobell are to be accounted for by the fact that the material examined by him was not satisfactory for a detailed study of the normal appearance of oöcysts. As *E. oxyspora* is in reality a coccidium of the testis of herrings, the heat employed in cooking the fish would account for any minute differences and the degenerate condition. Because many of the oöcysts seen by Snijders were in this condition, Brug (1922*a*) suggested that this coccidium was possibly not a human parasite, but an animal form which has been eaten with food. Brug's conjecture has been amply justified, and there is little doubt that, like *E. oxyspora*, *E. snijdersi* is merely *E. sardinæ*.

Brumpt (1918) states that the French armies were infected with " *Eimeria* " up to 0·2 to 0·33 per cent. He does not describe the organism, however, so it cannot be identified. Chatton (1918) records three cases of *Eimeria* infection in Tunis. He also does not describe the coccidia, but Mesnil (1919*a*) thinks they were probably *E. wenyoni* (*E. clupearum*).

EIMERIA IN OTHER MAMMALIA.

Pachinger (1886) described oöcysts of a coccidium from the kidneys of horses. The parasite was said to resemble *E. falciformis* of mice. Sampson (1917) stated that he had seen coccidia in the thickened mucosa of a horse which had become emaciated. From his meagre description it is impossible to tell if he was actually dealing with a coccidium, while the measurement he gives (3 mm.) is evidently incorrect. Selan and Vittorio (1924) state that they have found a coccidium named by them *E. utinensis* in the lungs and gall bladder of a horse in Italy. The description and figures are so unsatisfactory that it is impossible to form an opinion of the nature of the structures depicted.

Triffit (1924) has described as *E. canna* a parasite of the eland (*Orias canna*). The oöcysts measured 23·5 to 34 by 16·5 to 20 microns. It resembles the larger form found in cattle. Sheather (1923) noted the oöcysts of a coccidium, probably an *Eimeria*, in the fæces of an elephant. The writer and Scott (1925) discovered an *Eimeria* in the wallaby (*Macropus bennetti*). The usual forms occurred in the epithelium of the

intestine, while the oöcysts measured 22 to 34 by 10 to 17 microns and the sporocysts 7 to 11 by 6 to 8 microns. The name *E. macropodis* was given to the parasite.

EIMERIA IN BIRDS.

The first coccidium to be discovered in birds was one seen in fowls by Rivolta (1869). It was referred to by Eimer (1870). Rivolta (1873) described the parasite as *Psorospermi dei polli*, and to its presence he ascribed enteritis, laryngitis, rhinitis, stomatitis, conjunctivitis, and an inflammatory condition of the comb of chickens. In the same year Rivolta and Silvestrini (1873) wrote of it as *Psorospermium avium*. In his paper on the classification of these parasites, Rivolta (1878) named the organism *Gregarina avium intestinalis*, and stated that it occurred, not only in domestic Gallinaceæ, but also in the blackbird, crow, and other birds. It is evident that Rivolta was referring almost entirely to the oöcyst of the coccidia of fowls, which is an *Eimeria*, though he included the coccidia of the crow and other birds, which are now known to belong to the genus *Isospora*. Some observers, as, for instance, Reichenow (1921a), conclude that the name *P. avium* referred to an *Isospora*, but it is perfectly clear that Rivolta and Silvestrini gave their name to the parasite of chickens. Harz (1886) refers to the fowl parasite as *Coccidium rivolta*, a name which had been given by Grassi (1879) to an *Isospora* of the cat (p. 809).

Railliet and Lucet (1890a) described as *C. truncatum* a coccidium of the kidney tubules of geese. The oöcysts resembled those of *E. stiedæ*, but were smaller. It was included in the genus *Jarı ina* by Léger and Hesse (1922). Railliet and Lucet (1891) published an account of coccidiosis in fowls, and named the parasite *C. tenellum*, which name is often used for the chicken coccidium.

Eimeria avium (Rivolta and Silvestrini, 1873).—This is an intestinal coccidium of birds, and has been recorded in ducks, geese, chickens, pheasants, peacocks, turkeys, grouse, and other game birds (Fig. 350, 4). Hadley (1911) studied the infection especially in turkeys. Railliet and Lucet (1891a) stated that the infection was limited to the cæcum, but Hadley found that any part of the intestine, from the œsophagus to the rectum, might be involved, though the cæcum was the site of the heaviest infection. In large infections he noted that the parasites were not limited to the epithelial cells, but had spread to the subepithelial tissues, where large masses of reproducing organisms were found. The oöcyst, according to Hadley, varies very much in size, and may be as large as 38 by 29 microns or as small as 10·5 by 9 microns. The schizonts also vary in size from 10 by 8 to 58 by 32 microns. The largest schizonts produce over 300 merozoites, and the small ones from six to twenty. Intermediate forms also exist. The majority of observers have only noted the smaller schizonts, so that the possibility of two species occurring has to be considered. The oöcyst is ovoid, and has a micropyle at one end. It completes its development outside the body in two to three days at summer temperatures. There are produced the usual four sporocysts, each containing two sporozoites and a residual body (Fig. 350, 4).

The measurements in microns given by different observers for the oöcysts of *E. avium* from chickens, grouse, and turkeys vary considerably, as the following table shows:

		Oöcysts.	Sporocysts.
Railliet and Lucet, 1891 (chicken)	..	21–25 × 17–19	—
Fantham, 1910 (grouse)	.. ..	25–38 × 14–20	—
Hadley, 1911 (turkey)	.. ..	10·5–38·28 × 9–29·04	12 × 7
Jowett, 1911 (chicken)	.. ..	15–28 × 15–23	9–10 × 4–5
Gérard, 1913 (chicken)	.. ..	23–24 × 18–19	12 × 7
Reichenow, 1921 (chicken)	.. ..	15–20 × 13·5–18	10–11 × 5·5–6
Nieschulz, 1925 (chicken)	.. ..	14–27 × 12–22	—
Nieschulz, 1925 (turkey)	.. ..	17–31 × 14–24	—

Healthy birds are frequently found infected with this coccidium, but it is only in sick birds that very large infections occur. Whether in these cases the symptoms are entirely due to the coccidia or whether some other disease has lowered the resistance of the bird and enabled the coccidial infection to extend beyond the usual limits is difficult to state. Hadley at one time thought that the coccidia were responsible for a disease known as blackhead in turkeys, and Fantham that an epidemic with high mortality rate amongst young grouse in Scotland was due to the same organism. In both cases it is probable that the diseases were due to some other primary cause. Epidemics amongst newly hatched chickens are often the cause of serious mortality.

The great variations in the size of the oöcyst and other stages possibly indicate that there are more than one species of *Eimeria* in these birds, though Fantham (1910a) claims to have infected young chickens and pigeons by feeding them with fully developed oöcysts of the grouse coccidium.

Eimeria pfeifferi (Labbé, 1896).—This coccidium is a very common intestinal parasite of pigeons, and was named *Coccidium pfeifferi* by Labbé in 1896, but there has been considerable doubt as to whether it is distinct from *E. avium*. It has been studied by Nieschulz (1921a, 1921b, 1925a). The oöcysts measured from 15 to 26 microns in length by 14 to 24 microns in breadth. The sporocyst, which has one end pointed and the other rounded, measures on an average 12·5 by 6·5 microns. There is no evident micropyle in the oöcyst, and no residual body is formed within it, but a large one appears in each sporocyst. At the pointed end of the sporocyst there is a refractile knob. It will be seen that the measurements of the oöcysts agree very closely with those given by Nieschulz (1925a) for *E. avium* of the chicken. This observer finds, however, that the oöcysts of the pigeon coccidium tend to be spherical and those of the chicken coccidium ellipsoidal. Schizogony is of the usual type, and produces, as a rule, from fifteen to twenty merozoites, which vary in length from 5·5 to 9 microns. Nieschulz (1921b) attempted, without success, to infect chicks with the coccidium of the pigeon, though a control chick

acquired a heavy infection after ingesting a small number of oöcysts from a fowl. In further experiments (1925a) he succeeded in producing a mild infection in two of twenty chicks fed with numerous oöcysts. These two chicks later acquired a heavy infection of *E. avium* from chickens.

Jarrina paludosa Léger and Hesse, 1922.—Under the generic title *Jarrina*, Léger and Hesse (1922) described as *J. paludosa* a small coccidium of birds (*Fulica atra* and *Gallinula chloropus*). It resembles an *Eimeria*, but the oöcyst shows two peculiarities. The wall is punctate owing to fine canals which traverse its substance, while the end at which the micropyle is situated is drawn out like the neck of a bottle. The schizonts measured 10 microns in diameter, the microgametocytes 9 by 7
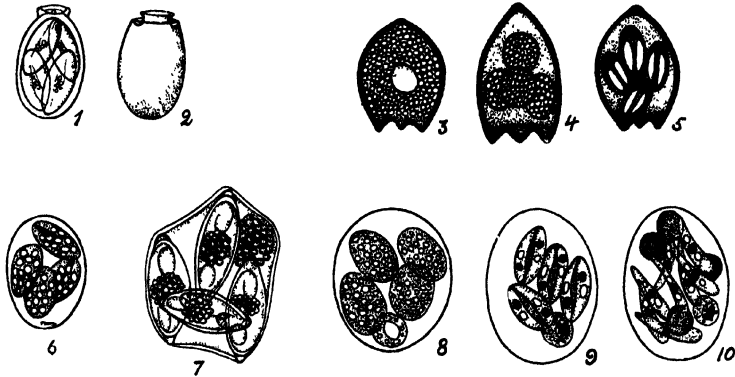
FIG. 369.—OÖCYSTS OF VARIOUS SPECIES OF *Eimeria* AND *Jarrina*. (1 AND 2, AFTER LÉGER AND HESSE, 1922; 7. AFTER DOBELL, 1908; OTHERS, AFTER LAVERAN AND MESNIL, 1902.)

     1-2. *Jarrina paludosa* from intestine of bird (× 1,200).
     3-5. *Eimeria mitraria* from intestine of tortoise, *Damonia reevesi* (× 1,100).
     6. *Eimeria ranarum* (× 1,000).   7. *Eimeria ranæ* (× ca. 1,000).
     8-10. *Eimeria (Paracoccidium) prevoti* (× 1,000).

microns, and the macrogametocytes 12 microns. The oöcysts, as seen in the fæces, measured 14 by 11 microns (Fig. 369, 1-2). Fifteen days at a temperature of 18° C. were required for complete development of the oöcyst. The sporocyst was equally pointed at each end, and measured 9 by 5 microns. Léger and Hesse believe that the small coccidium described by Labbé (1894) as *C. roscoviense* from the snipe and that recorded by Railliet and Lucet (1890a) as *C. truncatum* from the kidney of the goose may belong to this genus.

Lerche (1923) describes an *Eimeria* which develops in the kidney tubules of the goose in Germany. It often gives rise to a fatal infection. The oöcysts, which measure 14 to 22 by 11 to 15 microns, are rounded or ovoid in shape, and do not have the truncated end, as described by Railliet

and Lucet, though a slight flattening at the micropyle is sometimes seen. Sporogony resembles that of *E. stiedæ*, while schizogony occurs in the cells of the kidney tubules. It is possible that this form, which was also studied by Spiegl (1921), is *E. truncata*.

EIMERIA IN COLD-BLOODED VERTEBRATES.

Frogs and other cold-blooded vertebrates also harbour species of *Eimeria*. Frogs, in addition to the *Isospora* described above, are, according to Nöller (1920*b*), liable to infection with four species of *Eimeria*.

Eimeria ranarum (Labbé, 1894).—This coccidium was studied by Laveran and Mesnil (1902*b*). It is parasitic in the nuclei of the intestinal epithelium, and produces oöcysts measuring on an average 17 by 12 microns (Fig. 369, 6).

E. prevoti (Laveran and Mesnil, 1902).—This form is a parasite of the cytoplasm of the intestinal epithelium, and has oöcysts having average dimensions of 17 by 12 microns (Fig. 369, 8-10). It is remarkable that, after the formation of sporozoites in the usual manner, the sporocysts dissolve, leaving the eight sporozoites free in the oöcyst. For this reason Laveran and Mesnil (1902*b*) placed the parasite in a distinct genus (*Paracoccidium*). It appears, however, that dissolution of the sporocysts may occur in undoubted *Eimeria*, as, for instance, *E. falciformis* of mice.

E. ranæ Dobell, 1908.—The oöcysts of this form measure 18 by 22 microns. The four sporocysts are pointed at each end, and in this respect resemble the oöcysts of gregarines (Fig. 369, 7).

E. neglecta Nöller, 1920.—This species is a parasite of the intestine of the tadpole. It apparently does not occur in the adult frog. The oöcyst is spherical, and has a diameter of 9 to 10 microns.

Newts and salamanders also harbour coccidia belonging to this genus.

E. salamandræ (Steinhaus, 1889), with spherical oöcysts varying in diameter from 18 to 30 microns, develops in the nuclei of the cells of the intestine of the salamander. *E. propria* (Aimé Schneider, 1881) possesses oöcysts measuring 30 to 36 by 21 to 30 microns, and is an intestinal parasite of newts.

E. mitraria (Laveran and Mesnil, 1902).—This coccidium was discovered by Laveran and Mesnil (1902) in the intestine of the tortoise (*Damonia reevesi*). It is peculiar, not only in the character of the oöcysts, which are serrated at the end, but also in that the complete cycle of development appears to occur outside the cells in the lumen of the intestine (Fig. 369, 3-5).

Other species of *Eimeria* occur in reptiles, amphibia, and fish, and it is possible that most, if not all, vertebrates are liable to infection with these parasites. As noted above (p. 851) the oöcysts of *E. sardinæ* and *E. clupearum* of the herring may occur coprozoically in human fæces.

The following forms have been recorded:

SNAKES:

 E. cerastis Chatton, 1912 : *Cerastes vipera* and *C. cornutus* (oöcysts, 40 × 20 microns).

 E. sp. Phisalix, 1921: *C. cornutus* (oöcysts two sizes, 10 microns and 25 × 18 microns).

 E. crotali Phisalix, 1919: *Crotalus terrificus* (oöcysts, 32 × 22 microns).

 E. cystis-felleæ Debaisieux, 1914: *Tropidonotus natrix* (oöcysts, 30–38 × 20–25 microns).

 E. zamensis Phisalix. 1921: *Zamensis* sp. (oöcysts, 28–30 × 15–18 microns).

 E. tropidonoti Guyénot, Naville and Ponse, 1922: *T. natrix* (oöcysts, 20 × 10 microns).

 E. pythonis Triffitt, 1925: *Python molurus* and *P. sebæ* (oöcysts, 17–36 × 11·5–23 microns).

 E. sp. Grassi, 1888: *Coronella austriaca* (oöcysts, 14–15 microns).

LIZARDS:

 E. agamæ Laveran and Pettit, 1910: *Agama colonorum* (oöcysts 20–25 × 11–14 microns).

 E. scinci Phisalix, 1923: *Scincus officinalis* (oöcysts, 32 × 25 microns).

 E. raillieti Léger, 1899: *Anguis fragilis* (oöcysts, 18 microns in diameter).

 E. sp. Eimer, 1870; Danilewsky, 1896: *Lacerta* sp.

TORTOISE:

 E. mitraria Laveran and Mesnil, 1902: *Damonia reevesii* (oöcysts, 10–15 microns).

 E. legeri Simond 1901: *Emyda granosa* (macrogametes, 16–18 microns).

 E. delagei Labbé, 1893: *Emys orbicularis* (oöcysts, 25 microns).

CROCODILES:

 E. kermorganti Simond, 1901: *Gavialis gangeticus* (macrogametes, 20–22 microns).

 E. sp. Solger and Gabriel, 1876: *Crocodilus* sp.

FISH:

 E. subepithelialis Moroff and Fiebiger, 1905: Carp (oöcysts, 18–21 microns).

 E. rouxi Elmassian, 1909: Tench (oöcysts, 10 microns).

 E. gadi Fiebiger, 1913: *Gadus virens, G. morrhua, G. æglefinus* (oöcysts, 26–28 microns).

 E. percæ Dujaric de la Rivière, 1914: Perch (oöcysts, 12 microns).

 E. metchnikovi Laveran, 1897: *Gobio fluviatilis* (oöcysts, 20–25 microns).

 E. cotti Gauthier, 1921: *Cottus gobio* (oöcysts, 10–11 microns).

 E. piraudi Gauthier, 1921: *C. gobio* (oöcysts, 11–13 microns).

 E. truttæ Léger and Hesse, 1919: *Salmo fario* (oöcysts, 10–12 microns).

 E. alburni Stankovitch, 1920: *Cyprinus gobio* (oöcysts, 20 microns).

 E. cyprinorum Stankovitch, 1921: *Leuciscus rutilus, Scardinius erythrophthalmus, Barbus fluviatilis, Phoxinus lœvis* (oöcysts, 12–13 microns).

 E. cylindrospora Stankovitch, 1921: *Alburnus lucidus* (oöcysts, 10–11 microns).

 E. soufiæ Stankovitch, 1921: *Squalius agassizii* (oöcysts, 17–18 microns).

 E. anguillæ Léger and Hollande, 1922: Eel (oöcysts, 10 microns).

 E. cobitis Stankovitch, 1923: *Cobitis tænia* (oöcysts, 20 microns).

 E. misgurni Stankovitch, 1923: *Misgurnus fossilis, C. tænia* (oöcysts, 14–15 microns).

 E. sp. (*Goussia legeri*) Stankovitch, 1920: *A. lucidus, Scardinius erythrophthalmus, Abramis brama* (oöcysts, 10 microns).

 E. lucida Labbé, 1893: *Mustellus canis, Scyllium stellare, Acanthias acanthias* (oöcysts, 10–11 microns).

E. variabilis Thélohan, 1893, *Cottus bubalis*, *Gobius paganellus*, *Crenilabrus melops*, *Lepadogaster gouani* (oöcysts, 15–20 microns).

E. motellæ Labbé, 1893: *Motella tricirrata* (oöcysts, 13–14 microns).

E. cruciata Thélohan, 1892: *Trachurus trachurus* (oöcysts, 25 microns).

E. clupearum Thélohan, 1894 (syn. *E. wenyoni* Dobell, 1919): *Clupea pilchardus* (liver), *C. harengus* (liver), *Engraulis encrasicholus* (liver), *Scomber scomber* (oöcysts, 18–21 microns, except in intestine of *S. scombrus*, where they measured 35 microns).

E. minuta Thélohan, 1892: *Tinca tinca* (kidney, liver, spleen), (oöcysts, 9–10 microns).

E. thélohani Labbé, 1896: *Labrus* sp. (liver), (oöcysts, 25–30 microns).

E. bigemina Labbé, 1896: *Ammodytes tobianus* (oöcysts, 27–28 microns).

E. gasterostei Thélohan, 1890: *Gasterosteus aculeatus* (liver), (oöcysts, 16–18 microns).

E. sardinæ Thélohan, 1890 (syn. *E. oxyspora* Dobell, 1919): *Clupea pilchardus* (testis), (oöcysts, 40–50 microns).

E. gigantea (?) Labbé, 1896: *Lamna cornubica* (oöcysts, 70 microns).

E. wierzejski Hofor, 1904: *Cyprinus carpio* (oöcysts, 11–12 microns).

E. carpelli Léger and Stankovitch, 1921: *C. carpio* (oöcysts, 13–14 microns).

E. crystalloides (*Crystallospora crystalloides*) Thélohan, 1893: *Motella tricirrata*, *M. fusca* and *M. maculata* (oöcysts, 20–25 microns).

E. cyprini Plehn, 1924: *Cyprinus carpio*, *Tinca tinca* (oöcysts, 9 microns).

EIMERIA IN INVERTEBRATES.

The coccidium *E. schubergi*, which is parasitic in the centipede, *Lithobius forficatus*, has been described above. Another species, *E. lacazei*
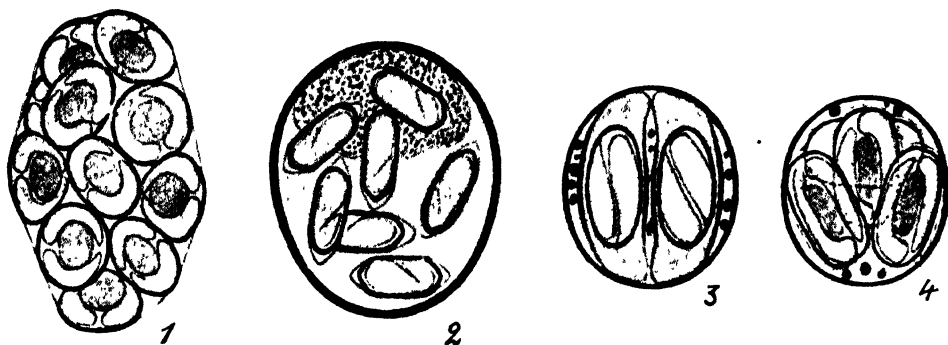
FIG. 370.—DIAGRAM OF THE OÖCYSTS OF THE FOUR COCCIDIA OF *Lithobius forficatus* (×ca. 1,000). (AFTER SCHELLACK AND REICHENOW, 1913.)

1. *Adelea ovata*.   2. *Barrouxia schneideri*.
3. *Eimeria lacazei*.   4. *Eimeria schubergi*.

In the case of 3, only two sporocysts are visible, as the other two are lying parallel to and beneath them.

Labbé, 1895, is more commonly found in this host. The oöcysts, which have a diameter of 22 to 25 microns, are almost spherical (Fig. 370, 3-4). The sporocysts are peculiar in that there is an elongate banana-shaped outer wall (epispore) and a much smaller oval inner wall (endospore).

There is a definite space between the two walls.   The space within the endospore is filled by the two sporozoites, which are closely packed together. On account of the character of the sporocyst, Labbé (1895) proposed placing the parasite in a distinct genus, *Bananella*.   It appears better, however, to retain it in the genus *Eimeria*.

Robertson, M. (1912) noted an interesting infection of a hemipteran (*Leptoglossus membranaceus*) in Uganda.   In this case the infection was not limited to the intestine, for oöcysts containing sporocysts and sporozoites occurred also in the salivary glands, while motile sickle-shaped individuals, probably merozoites, occurred in the proboscis.   As Robertson remarks, this parasite is of interest in indicating how an intestinal coccidium may eventually be inoculated to another host and acquire the habits of the hæmogregarines, which undoubtedly have arisen from intestinal coccidia.

Patton (1908*b*) observed a " small *Coccidium* " in *Lygæus militaris*.

(4) *Sub-Family* : BARROUXIINÆ.

This sub-family includes the two genera, *Barrouxia* Aimé Schneider, 1885, and *Echinospora* Léger, 1897.   In the former the oöcysts have a smooth outer surface, while in the latter a number of spines is present. The genus *Barrouxia* was founded by Aimé Schneider (1885) for a parasite of the water scorpion, *Nepa cinerea*.   It was named *B. ornata*.   Another form is *B. legeri* Schellack and Reichenow 1913, which was discovered in the centipede, *Lithobius impressus*, by Léger (1897), who identified it with *B. schneideri*.   Léger (1898) gave the name *B. caudata* to a form which is parasitic in another centipede, *L. martini*, and which is peculiar in producing a sporocyst provided with a tail-like prolongation (Fig. 372, 3-4). Awerinzew (1909*a*) described as *B. spiralis* a parasite of a worm (*Cerebratulus*).   The schizonts are peculiar in having a spiral form.   The best-known species of the genus is *B. schneideri*, which was called *Eimeria schneideri* by Bütschli (1882).   The genus *Echinospora* was founded by Léger (1897) for a parasite of the centipede, *L. mutabilis*.

Barrouxia schneideri (Bütschli, 1882).--The life-history of this parasite has been fully worked out by Schellack and Reichenow (1913).   According to them, it is the commonest of the four coccidia which occur in the intestine of the centipede, *Lithobius forficatus*.   Owing to the fact that mixed infections very commonly occur, various observers have confused stages of this parasite with others (Fig. 370).

During the process of schizogony the schizont increases in size, while the nuclei multiply by repeated divisions (Fig. 371).   The number of nuclei which are produced, and consequently the number of merozoites, varies considerably.   There may be as few as six or as many as twenty-six. Furthermore, the schizonts producing a small number of merozoites give

rise to smaller merozoites than those producing a larger number. When
merozoite production takes place, there may or may not be a residual
cytoplasmic body left over. The merozoites are elongate, slightly curved
bodies which in the schizont are often arranged like the segments of an
orange. The nucleus consists of a nuclear membrane, within which is a reticulum with a number of granules of chromatin. At one end of the nucleus is a definite karyosome. Though the merozoites differ in size and somewhat in shape, it was not possible to distinguish the merozoites which finally became gametocytes from those which developed into schizonts.

The growth of a macrogametocyte from a merozoite is recognized by its increase in size without nuclear multiplication. The nucleus enlarges and the cytoplasm becomes charged with food reserve material in the shape of
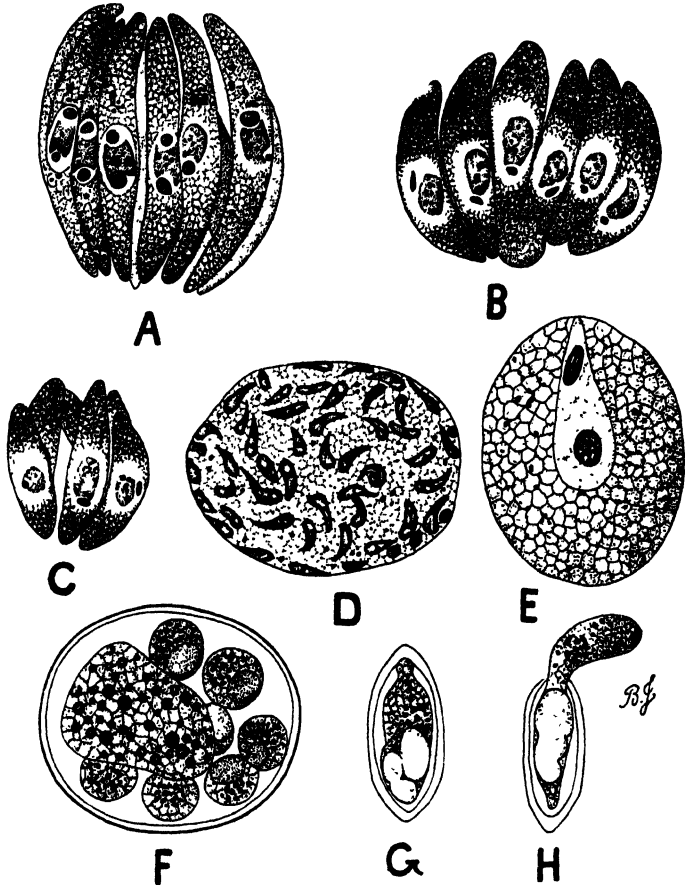
FIG. 371.—*Barrouxia schneideri* (× 1,650). (AFTER SCHELLACK
AND REICHENOW, 1913.)

A, B, C. Schizogony, showing varying number and size of merozoites.
    D. Microgametocyte with microgametes on surface.
    E. Macrogamete with elongated nucleus and microgamete nucleus
       (fertilization).
F Oöcyst with six sporoblasts and large residual body.
G. Sporocyst with single sporozoite.
H. Sporozoite escaping from sporocyst.

globules of a refractile substance. The nucleus retains its karyosome,
which is still present when the nucleus elongates at the time of fertiliza-
tion. The microgametocyte likewise develops from a merozoite, which
shows no special characteristics. The nucleus multiplies by repeated

divisions till a large number is formed. These nuclei collect on the surface and develop into microgametes.

After fertilization of the macrogamete an oöcyst is secreted and the nucleus again becomes spherical. Repeated nuclear divisions result in the formation of a number of nuclei, which approach the surface to become the nuclei of the sporoblasts, which are budded off, a large residual body being left. Each sporoblast becomes ovoid in shape and secretes around itself a sporocyst composed of two layers, an outer tough exospore and a thin endospore. The sporoblast within each sporocyst becomes a single sporozoite. The oöcyst is almost spherical, and measures on an average 30 by 26 microns (Fig. 370, 2). There is, however, considerable variation in size, for it may measure under 20 microns or over 40 microns in longest diameter. The number of sporocysts produced also varies within wide limits. There may be only three or as many as thirty. They measure 16 by 7 microns. The sporozoite escapes through a micropyle at one end of the sporocyst (Fig. 371, H).

From the descriptions of Aimé Schneider (1885, 1887), *B. ornata* has a similar life-history. Schneider considered, however, that the schizogony stages belonged to a distinct parasite, as it was not then realized that there occurred such an alternation of generations as is now known to exist in coccidia. The schizogony forms were called *Eimeria nepæ* and the oöcysts *Barrouxia ornata*. According to Schneider's figures, the oöcysts are spherical, and have a diameter of 34 to 37 microns. The oöcyst contains numerous sporocysts, each of which measures about 20 by 10 microns. The sporocyst, which contains a single sporozoite, is composed of two valves applied to one another

FIG. 372. — SPOROCYSTS OF COCCIDIA (× 1,500). (1 AND 3-4, AFTER LÉGER, 1898; 2, AFTER LABBÉ, 1896.)

1. *Echinospora labbei.*
2. *Eimeria crystalloides.*
3-4. *Barrouxia caudata*

like two watch-glasses. The valves finally separate to liberate the sporozoite.

Echinospora labbei Léger, 1897.—This coccidium is a parasite of the intestine of the centipede, *Lithobius mutabilis*, and was discovered by Léger (1897), according to whom (1897, 1897c, 1898) the oöcyst is spherical, has a diameter of 30 to 40 microns, and includes only four to eight sporocysts. Each is composed of two valves, measures 11 by 9·4 microns, and is peculiar in having a tuberculated surface (Fig. 372, 1).

<div align="center">(5) <i>Sub-Family</i> : CARYOSPORINÆ.</div>

This sub-family contains the single genus *Caryospora* Léger, 1904, which includes the single species *C. simplex*.

Caryospora simplex Léger, 1904.—This parasite was studied by Léger (1911), who described the main features of its development (Fig. 373). It occurs in the intestinal epithelium of the viper (*Vipera aspis*) of South Europe. There is a schizogony cycle in the epithelial cells, the fully grown schizonts measuring 8 to 10 microns in length and producing fifteen to twenty merozoites. After several cycles of asexual reproduction, micro- and macro-gametocytes are produced. Numerous microgametes are produced by the former, and the fertilized macrogamete becomes enclosed in a spherical oöcyst provided with a micropyle. The oöcyst is thick-walled, and has a diameter of 10 to 15 microns. The cytoplasmic contents of the oöcyst contract to form an ovoid body, round which a sporocyst provided with a micropyle is secreted. Within it are developed eight sporozoites and a residual body. This coccidium is remarkable in that only a single sporocyst is developed within the oöcyst. Léger (1911), from whose account this description is taken, has noted that in some cases the contents of the oöcyst break up into sporozoites without first forming a sporocyst.

<div align="center">(6) <i>Sub-Family</i> : PFEIFFERINELLINÆ.</div>

The members of this sub-family are characterized by the formation in the macrogamete of a " vaginal tube," an elongate cytoplasmic process by which the microgametes enter. The zygote produces an oöcyst in which eight sporozoites without sporocysts are formed. The sub-family includes the single genus *Pfeifferinella* Wasielewski, 1904, of which there are two species—*P. ellipsoïdes* Wasielewski, 1904, from the liver of the fresh-water mollusc, *Planorbis cornua;* and *P. impudica* Léger and Hollande, 1912, from the liver of the land snail, *Limax marginatus*.

Pfeifferinella impudica Léger and Hollande, 1912.—This parasite occurs in the liver cells of *L. marginatus* (Fig. 374). The schizonts have a diameter of 30 microns, and a large number of merozoites is produced.

These are peculiar in having one end tapering and mobile. There seems to be some indication of a membrane, and this, together with the presence of an axial filament arising from a granule near the nucleus, gives them the appearance of flagellates of the crithidia type. After schizogony has
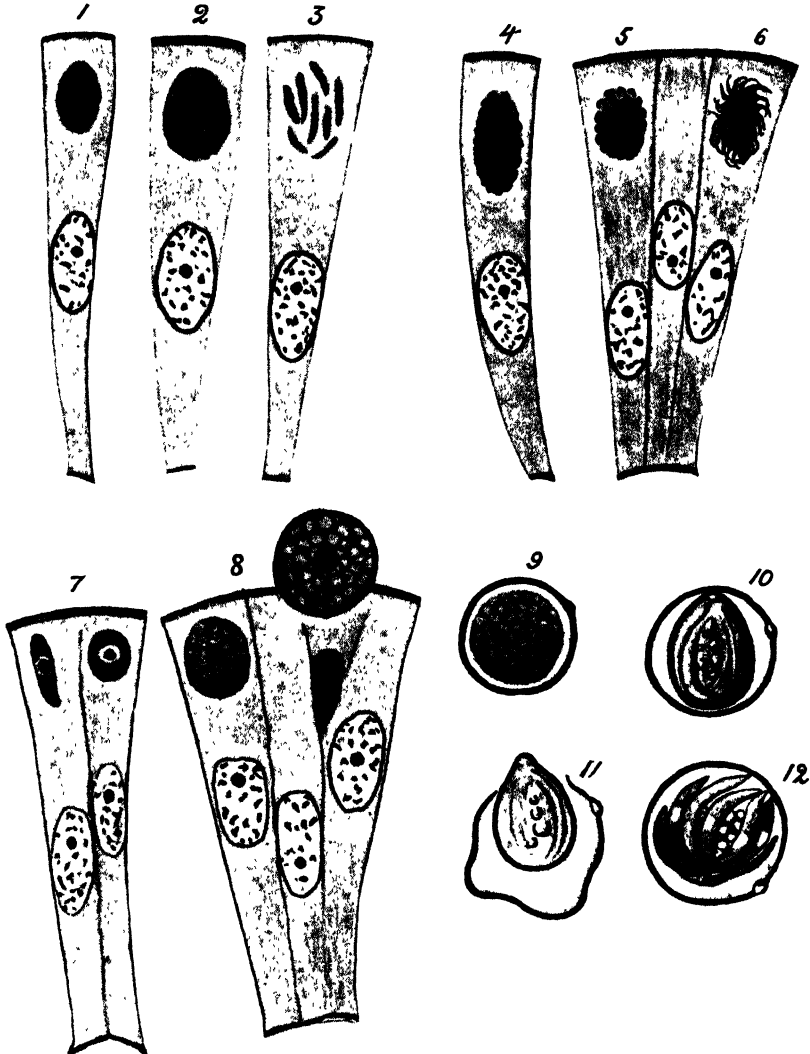
FIG. 373.—*Caryospora simplex* FROM THE INTESTINE OF THE VIPER (× 1,200). (AFTER LÉGER, 1911.)

1-3. Schizogony.
4-6. Development of male gametocyte and formation of microgametes.
7-8. Growth of female gametocyte.   9. Oöcyst containing zygote.
10. A single sporocyst has developed in the oöcyst.
11. Escape of sporocyst with eight sporozoites from the oöcyst.
12. Rupture of sporocyst before its escape from oöcyst.

been repeated several times, gametocytes are produced. The microgametocyte gives rise to a large number of very minute microgametes, while the macrogametocyte, just prior to fertilization, extrudes the remarkable process which has already been mentioned. The microgametes pass into the macrogamete along this process, which is subsequently retracted. The zygote becomes enclosed in an ovoid oöcyst measuring 20 by 10 microns. In it are produced eight sporozoites and a residual body.
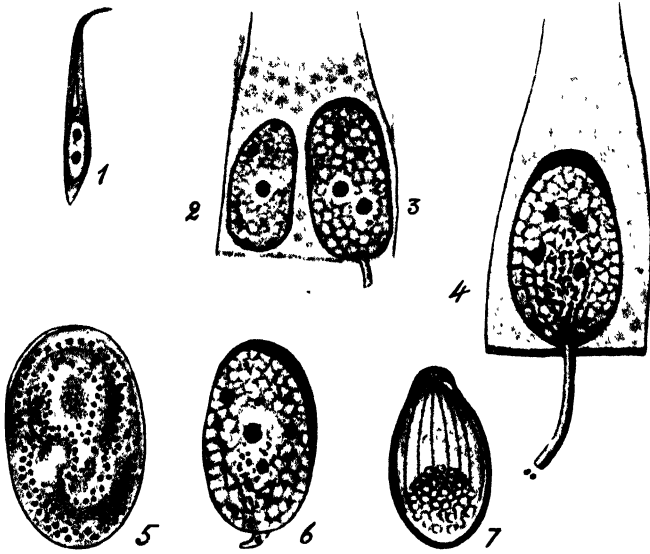
FIG. 374.—*Pfeifferinella impudica* (× 1,200). (AFTER LÉGER, L., AND HOLLANDE, 1912.)

1. Merozoite, showing crithidia-like structure. 2-3. Growing macrogametocytes.
4. Mature macrogametocyte with fertilization process, at apex of which are two microgametes.
5. Microgametes forming in microgametocyte.
6. Macrogamete withdrawing fertilization process after entry of male gamete.
7. Oöcyst with eight sporozoites.

4. *Family :* CARYOTROPHIDÆ Lühe, 1906.

Of this family there is only a single genus, *Caryotropha* Siedlecki, 1902, and one species, *C. mesnili* Siedlecki, 1902, which is parasitic in the body cavity of a marine worm, *Polymnia nebulosa*.

Caryotropha mesnili Siedlecki, 1902.—The development of this parasite was studied by Siedlecki (1902, 1907), and is of interest in that it varies from the type which is usual in the more typical coccidia (Fig. 375). The merozoite, as presumably did the sporozoite, enters a cell of the body cavity or spermatogonium, where it grows into a large schizont. The latter, however, does not divide into merozoites directly, but produces a number of intermediate bodies (meroblasts), which are separated from one another
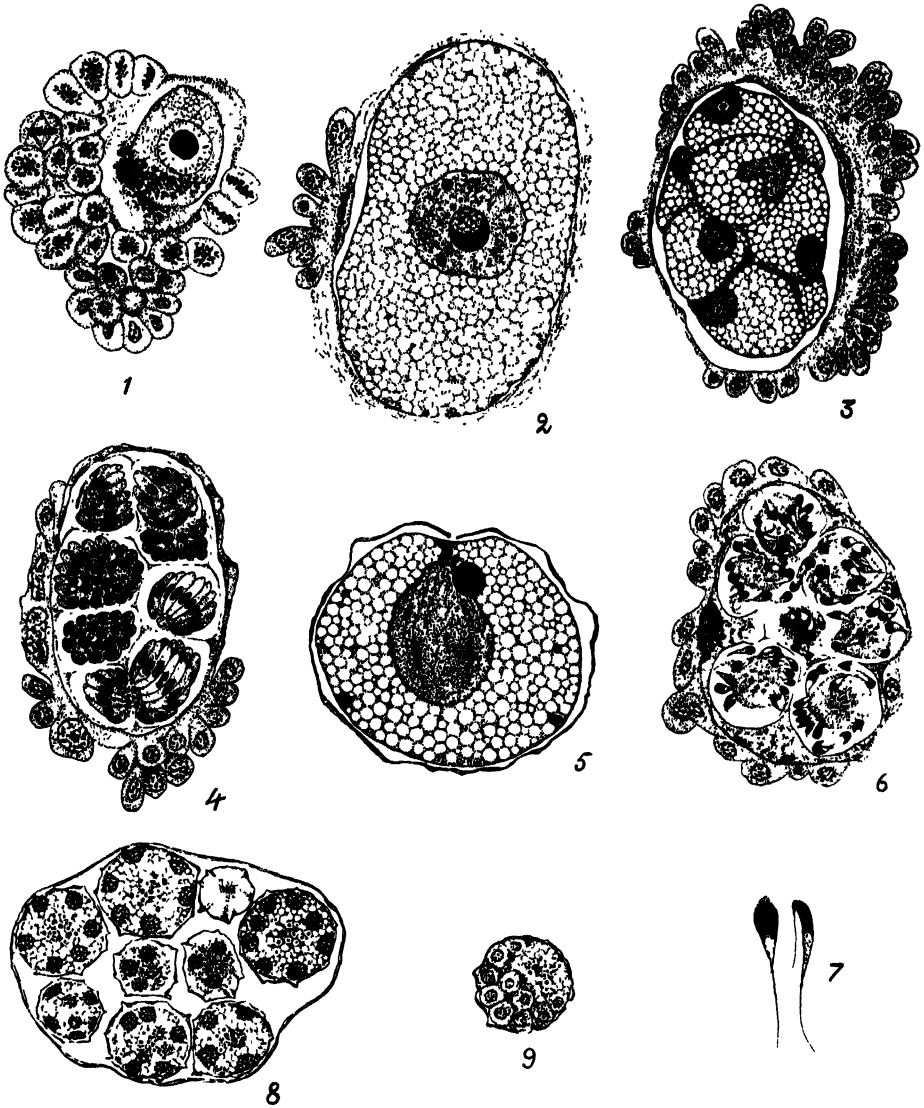
FIG. 375.—STAGES IN DEVELOPMENT OF *Caryotropha mesnili* PARASITIC IN THE BODY CAVITY OF THE MARINE WORM, *Polymnia nebulosa* (× 630). (AFTER SIEDLECKI, 1907.)

1. Partially grown schizont in mass of spermatogonia.
2. Fully grown schizont.
3. Schizont segmented into cytomeres.
4. Each cytomere has produced a number of merozoites.
5. Macrogametocyte in section. Escape of karyosome from the nucleus.
6. Microgametes formed from the separate bodies into which the microgametocyte divides.
7. Individual microgametes, showing flagella at double magnification.
8. Oöcyst containing a number of sporocysts. The nucleus of the sporoblasts has multiplied.
9. Single ripe sporocyst with sporozoites and residual body.

by septa representing the remains of the parent. Each meroblast then divides into a number of merozoites, each of which can enter a new cell and repeat the process. Some merozoites now develop into macro- and micro-gametocytes. The former, as in other coccidia, becomes a macro-gamete, but the latter, instead of producing microgametes directly, divides into a number of intermediate bodies (microgametoblasts) which, as in the case of the meroblasts, lie in spaces in the remains of the micro-gametocyte, where they each give rise to a number of microgametes. The macrogamete after fertilization becomes enclosed in a spherical oöcyst provided with a micropyle. Within it are produced about twenty spherical sporocysts, each of which contains twelve sporozoites.

5. *Family:* AGGREGATIDÆ Labbé, 1899.

This family contains the genus *Aggregata* Frenzel, 1885, of which there are, according to Moroff (1908), over twenty species, and several other genera which possibly should be included here. As the members of the genus *Aggregata* are peculiar in that the schizogony cycle occurs in crabs and the sporogony cycle with the production of oöcysts in cephalopods, it was not at first realized that the forms in these two hosts were merely stages of one parasite. The parasite of the cuttlefish, *Sepia officinalis*, was first recorded by Lieberkühn (1854). He included it under the general name Psorosperm, but later (1855) classed it with the gregarines as *Monocystis*. Aimé Schneider (1875*a*) gave the name *Benedenia octopiana* to a form in *Octopus*, while later (1883) he placed the parasites of *Octopus* and *Sepia* in his genus *Klossia* as *K. octopiana*. Labbé (1895*a*) gave the name *B. eberthi*, changed to *K. eberthi* by him (1896), to a form with sporocyst having three or four sporozoites. Other generic titles were employed, such as *Legeria*, by Blanchard (1900), *Eucoccidium* by Lühe (1902), and *Legerina* by Jacquemet (1903). The stages in crabs seem to have been noted long before those in cuttlefish, for Labbé (1899) states that they were seen by Redi (1708), Cavolini (1787), Rudolphi (1819), and Diesing (1851). Frenzel (1885) gave the name *Aggregata* to several forms, and, Schneider's name, *Benedenia*, being preoccupied (Diesing, 1858), this is undoubtedly the correct name of the genus.

The first observers to recognize the fact that the parasites of the crab and cephalopod were different stages of one organism were Léger and Duboscq (1906). Siedlecki (1898) had described the development in the cephalopod, and come to the conclusion that the development and method of fertilization was of the coccidium type, and that the parasite was a coccidium and not a gregarine, as had been supposed. Moroff (1906*a*), however, denied this, and claimed that there occurred a union of gametes, as in gregarines. As a result of his statement, Léger and Duboscq (1908) placed the parasites amongst the schizogregarines in a separate family (*Aggregatidæ*). Moroff (1908), however, modified his former opinion, and described the process of microgamete formation of the parasite in the crab as being like that of a coccidium. The investigations of Dobell (1914, 1925) and Pixell-Goodrich (1914) confirmed the latter observations, and finally proved the correctness of the statements made by Siedlecki in 1898, so that the *Aggregatidæ* may be safely regarded as coccidia, which have their schizogony stage in one host and the sporogony in another.

The form which has been most completely studied is the one which was named *Klossia eberthi* by Labbé (1896). Dobell (1925) has recently given a complete account of its life-history.
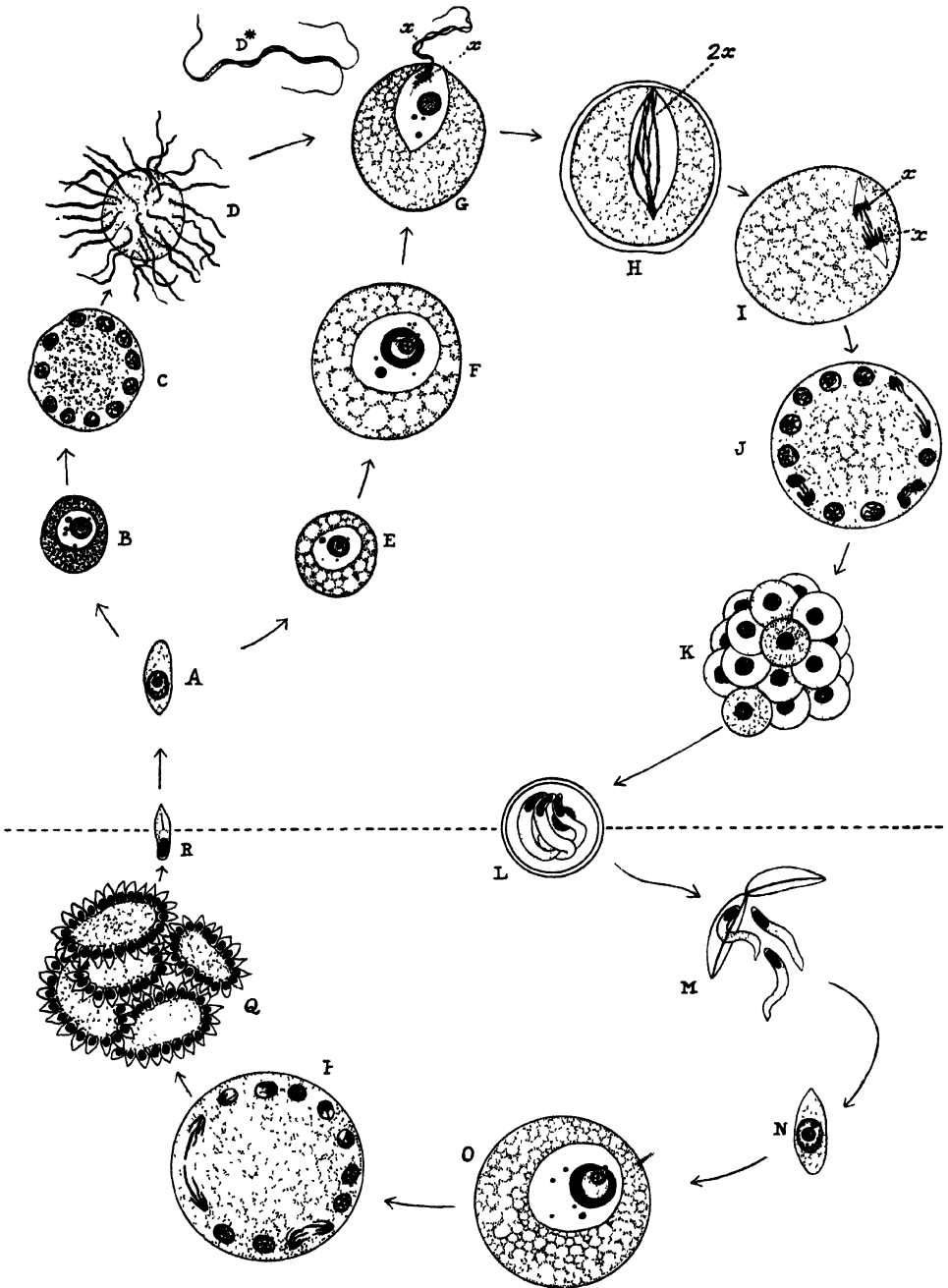
FIG. 376.—DIAGRAM REPRESENTING LIFE-CYCLE OF *Aggregata eberthi :* THE STAGES
ABOVE THE DOTTED LINE OCCUR IN THE CUTTLEFISH (*Sepia officinalis*), THOSE
BELOW IN THE CRAB (*Portunus*).   (AFTER DOBELL, 1925.)

[*For description see opposite page.*

Aggregata eberthi (Labbé, 1895).—This parasite, which is the best-known member of the genus, passes its schizogony cycle in the crabs, *Portunus depurator* and *P. armatus*, and its sporogony cycle in the cephalopod, *Sepia officinalis* (Fig. 376). The cycle in the crab has been studied by Léger and Duboscq (1908) and Dobell (1925). The sporocysts, taken into the intestine of a crab which has eaten infected material passed in the dejecta of a cuttlefish, liberates its three sporozoites by separation of the two valves of which it is composed (Fig. 377). The sporozoites then pass through the lining cells of the gut and settle down in the peri-intestinal connective tissue. Here they grow enormously in size during the next thirty or forty days, and are differentiated, according to Léger and Duboscq, into male forms with a thick enclosing membrane and female forms with a thin membrane. The latter, which may reach a diameter of 200 microns, are larger and grow more quickly than the male forms. They are termed male and female forms, because it is supposed the merozoites derived from them, when taken up by a cuttlefish, give rise to male and female gametocytes respectively. Dobell (1925), however, has found that no such sexual dimorphism of the schizonts occurs. The nucleus of these adult forms is remarkable. A female form of 100 microns in diameter had a nucleus measuring 60 microns, which enclosed a nucleolus of 23 microns. When fully grown, the schizonts prepare for schizogony by nuclear multiplication. Remarkable changes occur in the large nucleus, which moves towards the surface. Its nuclear membrane is lost, and the chromatin is liberated into the cytoplasm. Some of the chromatin becomes arranged as chromosomes, which, according to Dobell and Jameson (1915), are six in number, and form a series from a long one to a short one. A form of mitosis (promotosis), in which each chromosome divides, gives rise to two groups of six, each of which groups constitutes the chromatin of a daughter nucleus. Before, however, the daughter nuclei are actually constituted, two new spindles appear in association with the daughter groups of chromosomes, which become arranged on the equator of the spindles and divided as in the first division. By a repetition of this process, in which each succeeding division commences before definite

R. Merozoite swallowed by the cuttlefish.
A. Undifferentiated parasite in submucous tissue.
B-D. Growth into microgametocyte and production of microgametes.
E-G. Growth into macrogametocyte and fertilization.
H. Zygote, the nucleus of which contains twelve chromosomes, six derived from the microgamete and six from the macrogamete ($2x$).
I. First nuclear division in zygote; the twelve chromosomes form two groups of six, so that each resulting nucleus has six chromosomes (x).
J-K. Nuclear multiplication in zygote and production of sporoblasts.
L. Sporocyst containing three sporozoites and small residual body.
M. Escape of sporozoites in intestine of crab.
N-Q. Growth of schizont and production of merozoites in subepithelial connective tissue.

nuclei are formed, a large number of groups of six chromosomes is formed on the surface of the schizont. After the last division definite nuclei are formed, consisting of a nuclear membrane and granules of chromatin, into which the six chromosomes of each group have broken up. Meanwhile, the cytoplasm of the schizont becomes lobulated and vacuolated so as to form a coarse cytoplasmic meshwork. This process bears a striking resemblance to that which takes place in the development of the zygotes
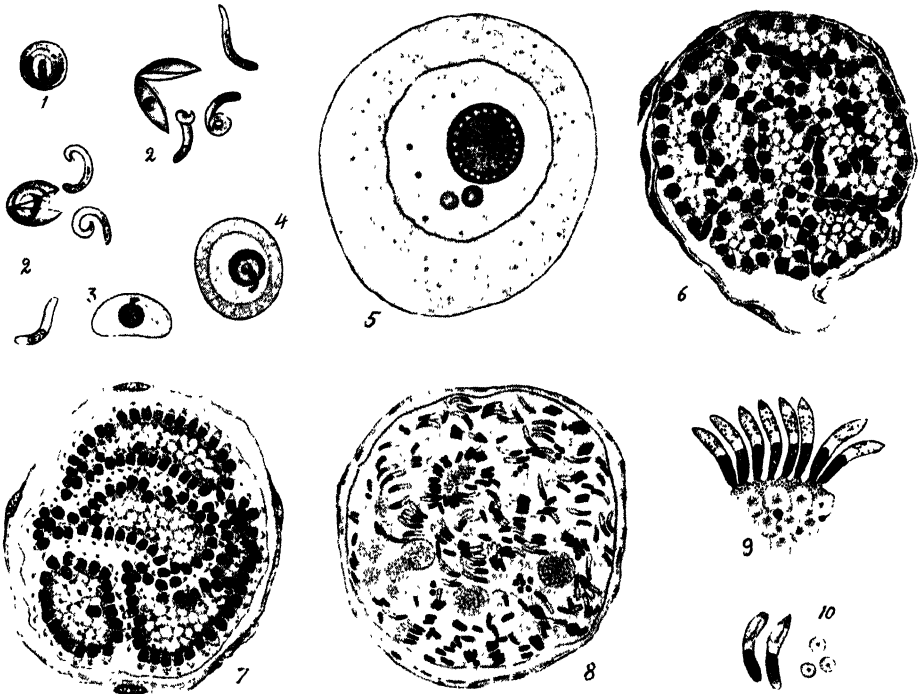
FIG. 377.—SCHIZOGONY OF *Aggregata eberthi* IN THE INTESTINE OF THE CRAB (*Portunus*). (AFTER LÉGER AND DUBOSCQ, 1908.)

1. Oöcyst with three sporozoites (× 1,000). 2. Escape of sporozoites (× 1,000).
3-5. Growth of schizont (× 1,000). 6. Multinucleated schizont—male (× 850)
7. Production of merozoites—male (× 850).
8. Completion of schizogony—female (× 850).
9. Merozoites formed as finger-like outgrowths and still attached to residual cytoplasm—male (× 850).
10. Merozoites, showing axial fibre in side view and in cross-section—female.

of malarial parasites in the stomach of mosquitoes, and seems to be a means of increasing the surface area of the cytoplasm on which the nuclei arrange themselves. The method by which the merozoites are formed is an indication of this necessity. Opposite each nucleus the cytoplasm becomes elevated and grows out as a finger-like process, which takes the nucleus with it (Fig. 377, 7). This growth continues till the merozoite

attains the required length, and, as merozoites are being formed simultaneously on every surface, the cytoplasm is gradually absorbed into the outgrowing merozoites, which are still attached to the remains of the cytoplasm (Fig. 377, 9). The latter, which at first is a continuous branching network, becomes broken up into irregular masses, each of which has merozoites attached in a radiating manner. Léger and Duboscq claimed that the merozoites formed from the smaller female schizonts are of the same length, but broader, and have larger nuclei than those derived from the larger male schizonts. Furthermore, the merozoites of the female line have a well-marked axial filament (Fig. 377, 10). As in the case of the schizonts, Dobell could not confirm these statements regarding a sexual dimorphism of the merozoites, which appeared to him to be all of one type. No further development occurs in the crab, and it is assumed that the cuttlefish become infected by eating the crabs, the merozoites penetrating the gut wall and developing into male and female gametocytes of large size. The nucleus of the microgametocyte multiplies, and finally a number of long microgametes, each with two anterior flagella, is formed. The macrogamete, the nucleus of which has undergone remarkable changes, is fertilized by a microgamete. The nucleus of the resulting zygote multiplies by repeated divisions, the first of which is a reducing division, as explained above (p. 109). Eventually there is produced a number of sporoblasts. The latter become sporocysts, which are usually fusiform bodies pointed at each end. Sometimes, however, they are spherical. They are, as a rule, about 9 microns in length, but may be only 4 microns, or as much as 25 microns. Within each sporocyst are developed three sporozoites and a residual body (Fig. 376, L). During its development the sporont is lying in the tissues of the gut wall of the cuttlefish enclosed in a membrane derived from the tissues of the host. There seems to be some doubt as to whether a distinct oöcyst formed by the parasite is present or not. The sporocysts, which escape in the faeces of the cuttlefish, are eventually eaten by crabs, and in them the sporozoites recommence the asexual cycle.

The nucleus of *Aggregata eberthi* has been the subject of a detailed study by Dobell (1925). When the merozoite or sporozoite commences to grow, there soon appear in the nuclei two structures which could not be detected in the earliest stages. One of these Dobell calls the karyosome and the other the " micronucleus." Both of these are present in the nuclei of all stages of development. As growth of a nucleus takes place, its karyosome becomes larger and finally hollow. Through a pore (micropyle) which is formed the micronucleus passes to occupy a position within the karyosome. The karyosome may bud off one or more secondary karyosomes, while the chromatin granules, which in the earlier stages of

growth occurred within the nuclear membrane, disappear. The structure
of a typical nucleus is shown in the diagram (Fig. 378). When a nucleus is
about to divide, the "micronucleus," or the substance into which it breaks
up, passes through the pore in the karyosome. The nuclear membrane
disappears and the karyosome gradually disintegrates. Meanwhile, chromo-
somes are developed from the "micronucleus" and mitotic division occurs.
The term micronucleus was employed for the body within the karyosome,
because the chromosomes were formed from it alone, and because it gave
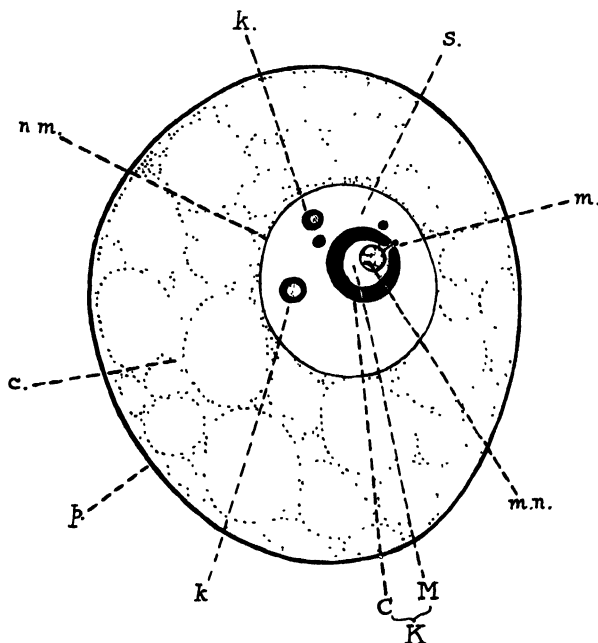
FIG. 378.—DIAGRAMMATIC REPRESENTATION OF THE STRUCTURE OF *Aggregata
eberthi* (♀). (AFTER DOBELL. 1925.)

c., Cytoplasm; *p.*, pellicle; *n.m.*, nuclear membrane; *s.*, nuclear space; *K.*, main karyosome;
C., cortex of karyosome; *m.*, micropyle of karyosome; *M.*, medulla of karyosome; *m n.*, micro-
nucleus within karyosome; *k.*, accessory karyosomes which are formed by budding from
main karyosome.

rise to the daughter nuclei. According to Dobell, the small nucleus
(micronucleus) is included within the large nucleus, the two structures
being comparable with the micronuclei and macronuclei of ciliates.
Bělař (1926), however, doubts if Dobell's interpretation is correct. He
does not regard the "micronucleus" as a true nucleus, for he finds that
the chromosomes may be already present when the "micronucleus" is
intact. Moreover, he claims that several "micronuclei" may occur in one
nucleus and to have demonstrated centrosomes which were not seen by
Dobell.

Other species of *Aggregata* which are also parasitic in crabs and cephalopods are *A. octopiana* (Aimé Schneider, 1875), which has sporocysts containing ten to twelve sporozoites; *A. spinosa* Moroff, 1908, the sporocysts of which are provided with spines and contain numerous sporozoites; *A. legeri* Moroff, 1908, with sporocysts having sixteen sporozoites; and *A. duboscqi* Moroff, 1908, having sporocysts with eight sporozoites. According to Moroff (1908), there are over twenty known species, but many of these, according to Dobell (1925), are not valid.

There are several genera, which probably belong to this family, the life-cycles of the members of which are not completely known. In those stages which have been studied there is a resemblance to species of *Aggregata*.

The genus *Pseudoklossia* was founded by Léger and Duboscq (1915a) for a parasite of lamellibranch molluscs (*Tapes floridus* and *T. virgineus*). It is found chiefly in the kidneys. The schizogony cycle could not be discovered, and this led the authors to conjecture that, as in species of *Aggregata*, it takes place in some other host. The parasite, which was named *P. glomerata*, has a fertilization process of the *Eimeria* type. The oöcysts are about 40 microns in diameter, and contain numerous spherical sporocysts 4 to 5 microns in diameter. Each sporocyst contains two sporozoites. Another species (*P. pectinis*) was described by the same authors (1917) from the kidneys of *Pecten maximus*, while Debaisieux (1919) recorded two new species, *P. chitonis* and *P. patellæ*, from *Acanthochites fascicularis* and *Patella vulgaris*. According to Léger and Duboscq (1915a), it is probable that the parasite described by Léger (1897b) as *Hyaloklossia pelseneeri*, and which is parasitic in species of *Tellina* and *Donax*, belongs to this genus.

A closely allied genus is *Merocystis*, which was established by Dakin (1911) for a parasite of the kidneys of the whelk (*Buccinium undatum*). The oöcysts are polysporocystid and the sporocysts dizoic, as in members of the genus *Pseudoklossia*. The development of the microgametocyte differs, however, in *Merocystis kathæ*, the only known species, in that it segments into a number of cytomeres, each of which gives rise to a large number of gametes, as in the case of *Caryotropha mesnili*, considered above. The parasite was later studied by Foulon (1919), who pointed out its resemblance to the sporogony cycle of species of *Aggregata*. Another genus, *Myriaspora*, was established by Lermantoff (1913) for a coccidium of an annelid, *Trophonia plumosa*, which he named *Myriospora trophoniæ*. The sporogony cycle is similar to that of *Merocystis kathæ*. The macrogametocyte grows as a vermicular body till it reaches a size of 700 to 800 microns in length by 100 microns in breadth. The microgametocyte is also vermicular, and when it has reached a length of 200 microns is curved like a horseshoe. It then divides into about 100 cytomeres, which give rise to microgametes. The oöcyst, which is formed after retraction of the macrogamete, contains some hundreds of sporocysts, each of which has from twenty-four to thirty-six sporozoites. The genus *Angeiocystis*, which was founded by Brasil (1904) for a single species, *A. audoniniæ*, parasitic in a worm (*Audoninia tentaculata*), resembles the preceding one. Both the macrogametocyte and microgametocyte are vermicular. The microgametocyte apparently gives rise to microgametes directly without the formation of cytomeres. The oöcyst has four sporocysts, each of which measures 22 by 15 microns and contains about thirty sporozoites.

Duboscq (1917) founded the genus *Selysina* for a curious parasite of the ascidian *Stoloica socialis*. He named it *S. perforans*. In a later paper (1918), he described

various stages of the organism which occurred in the larval forms as well as the adult ascidians. In addition to unencysted stages, which occurred in what appeared to be very much hypertrophied leucocytes, there were sporocysts measuring 15 by 5 microns, and containing each a single sporozoite, as also large cysts up to 500 microns in diameter. The latter had walls as much as 50 microns in thickness and were filled with bunches of merozoites.

6. *Family:* LANKESTERELLIDÆ Reichenow, 1921.

The forms included in this family have been long recognized as hæmo-gregarines of cold-blooded animals. The researches of Reichenow and Nöller have shown that the forms within the red blood-corpuscles are sporozoites, and that the remainder of the cycle takes place in the intestinal wall or blood-vessels, and is of the *Eimeria* type. According to the cycle of development, two sub-families can be recognized.

(1) *Sub-Family :* SCHELLACKIINÆ.

In this sub-family there is a single genus, *Schellackia*, founded by Reichenow (1919). The whole of the development takes place in the wall of the intestine, the sporozoites finally entering the blood-corpuscles.

Schellackia bolivari Reichenow, 1919.—This parasite is the best-known member of the genus, and has a cycle of development which closely corresponds with that of *E. schubergi* (Fig. 379). It is found in the intestinal epithelium of the mid-gut of the lizards, *Acanthodactylus vulgaris* and *Psammodromus hispanicus*, in which the schizogony cycle, with production of ten to sixteen merozoites, takes place. Certain merozoites eventually develop into micro- and macro-gametocytes. The merozoites which produce the former develop in the epithelial cells as usual, and eventually produce a large number of biflagellate microgametes. The merozoites which produce macrogametocytes, however, wander through the epithelial cells and develop in the subepithelial connective tissue, and it is here that fertilization takes place and the oöcyst is formed. Within the oöcyst the zygote breaks up into eight sporozoites and a residual body. As the ripe oöcyst is formed in the subepithelial connective tissue, it is evident it cannot readily escape into the lumen of the gut to be voided in

1. Entry of sporozoite into intestinal cell of lizard.
2-5. Schizogony cycle in intestinal cells.
6-9. Growth of microgametocyte and formation of microgametes in intestinal epithelial cells.
10-11. Growth of macrogametocyte in subepithelial tissue.
　12. Fertilization of macrogamete.
13-16. Growth of zygote and sporogony producing sporozoites.
　17. Entry of sporozoite into blood-vessel of lizard.
　18. Sporozoites within the red blood-corpuscles.
　19. Red blood-corpuscles containing sporozoite being phagocyted by intestinal epithelial cell of mite
　20. Red blood-cells within intestinal epithelial cells of mite.
　21. Group of sporozoites which have escaped from the digested red blood-corpuscles. When the mite is devoured by the lizard, the sporozoites make their way into the intestinal epithelial cells of the lizard and the schizogony cycle is commenced.
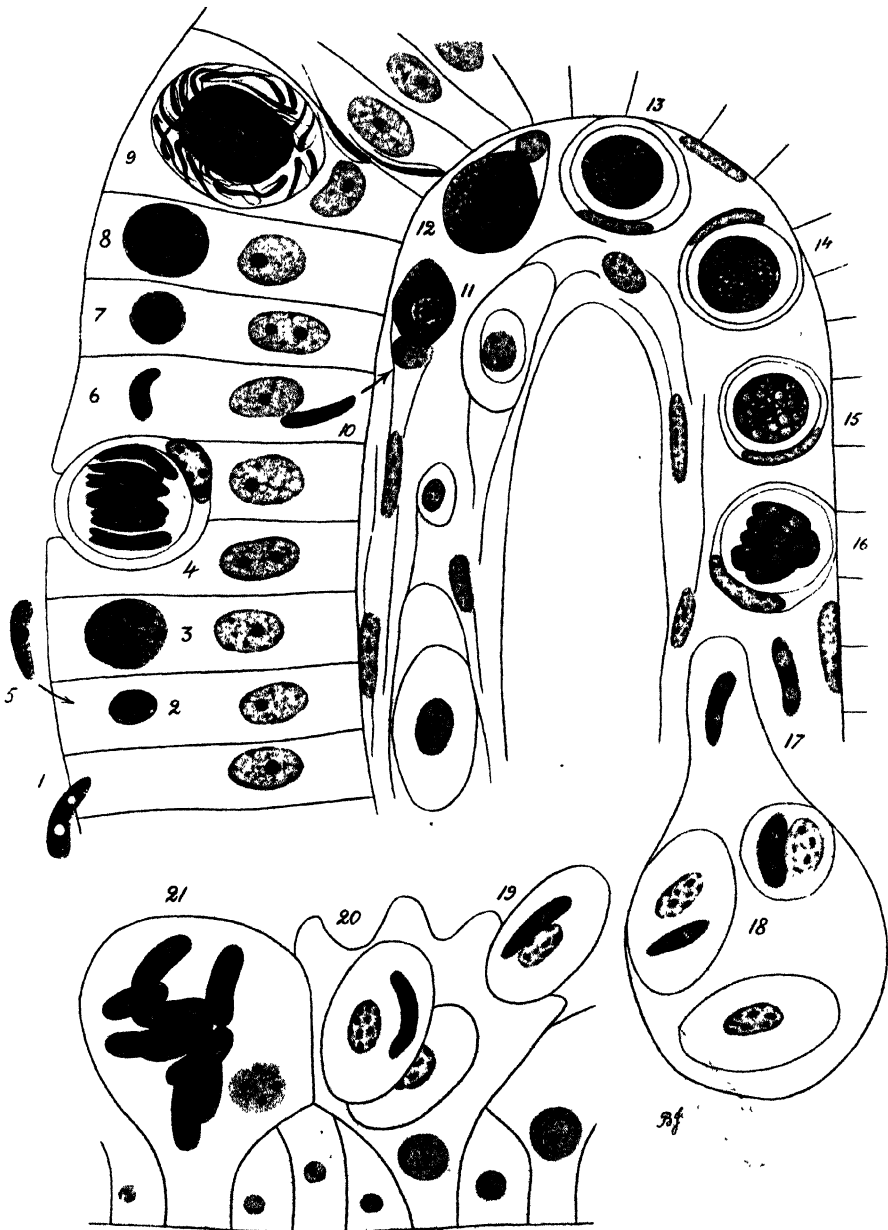
FIG. 379.—LIFE-CYCLE OF *Schellackia bolivari* IN THE LIZARD, *Acanthodactylus vulgaris*, AND THE MITE, *Liponyssus saurarum* (× ca. 1,000). (AFTER REICHENOW, 1921.)

the fæces, as occurs in the typical intestinal coccidia. Another course is adopted, and in it, as in that which is followed by members of the succeeding sub-family, there is to be noted the gradual adaptation of an intestinal coccidium to a blood habitat. The ripe oöcyst in the subepithelial connective tissue ruptures, the sporozoites are liberated and penetrate the blood-corpuscles. Here they appear in the circulation as hæmogregarines. In the case of *A. vulgaris* it is the red blood-corpuscles which are invaded by the sporozoites, whereas in *P. hispanicus* it is the lymphocyte which is nearly always infected. Reichenow demonstrated this fact by cross-infection experiments. It is evident, therefore, that a parasite may occur in one type of cell in one host and in a different type of cell in another. Within these cells they remain unchanged till the blood is taken up by the lizard mite, *Liponyssus saurarum* (Fig. 458). In the intestine of the mite the phagocytic activities of the gut epithelial cells lead them to engulf infected red cells, which are digested, so that the contained sporozoites (hæmogregarines) become free in the cytoplasm. In this manner groups of sporozoites collect in the cells, where they increase in size to a slight extent, but undergo no further change. When the mite is eaten by the lizard, as frequently happens, the sporozoites escape into the lizard's gut, infect the epithelial cells, and recommence the schizogony cycle.

Two other species of *Schellackia* have been studied by Reichenow in *Lacerta muralis*, which may harbour as many as nine species of hæmogregarine.

<center>(2) <i>Sub-Family :</i> LANKESTERELLINÆ.</center>

This sub-family contains the single genus, *Lankesterella*. The whole of the development takes place in the endothelial cells of the blood-vessels, the sporozoites finally entering the blood-corpuscles. The genus was founded by Labbé (1899) for a little parasite of the red blood-corpuscles of the frog, which was first seen by Chaussat (1850).

Lankesterella minima (Chaussat, 1850).—This parasite has been studied chiefly by Nöller (1912b, 1913a, 1913b, 1920b), who gives the following synonyms: *Anguillula minima* Chaussat, 1850; *Drepanidium ranarum* Lankester, 1871, 1892; "Würmchen" Gaule, 1880, 1881, 1886; "Cytozoon" Bütschli, 1882; *D. ranarum* (Lankester) Wallerstein, 1882; *D. princeps* Labbé, 1894; "Drepanidio piccolo" pro parte Grassi and Feletti, 1892; *L. ranarum* Labbé, 1899; *Hæmogregarina minima* (Laveran) Mathis and Léger, 1911.

As seen in the red blood-corpuscles of the frog, *L. minima* is a small vermicule, which may reach half the length of the corpuscle, but not more. Many of the forms are smaller than this. Various observers have described the vermicule as becoming spherical and reproducing in the red cells by schizogony, but Nöller (1913b) believes the schizonts belong to *Dactylosoma ranarum*, another parasite of the frog's corpuscles (p. 1039).
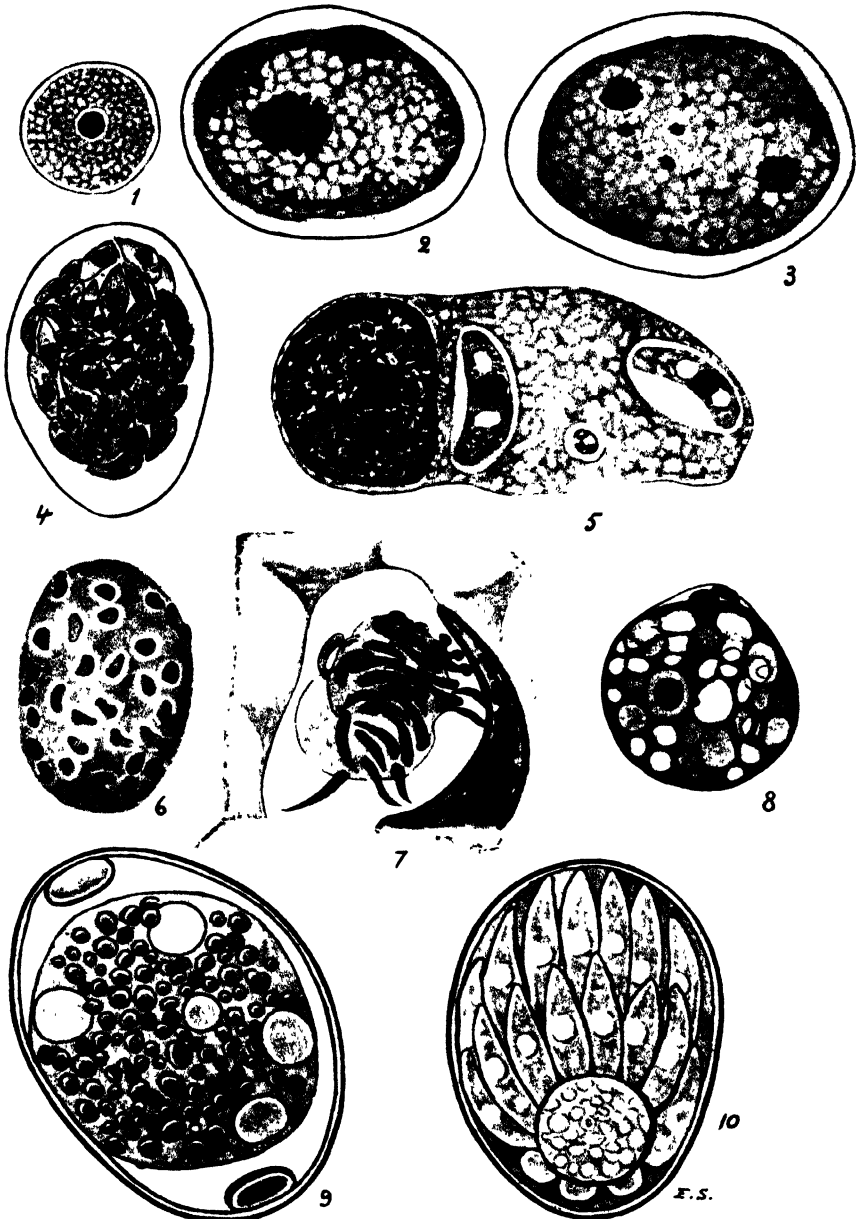
FIG. 380.—DEVELOPMENT OF *Lankesterella minima* IN THE FROG (1-5, × 2,700; 6-10, × 1,500). (AFTER NÖLLER, 1913 AND 1920.)

1-4. Stages in schizogony in the endothelial cells of the vessels of the organs. This is initiated by sporozoites inoculated by the leech. 5. Young forms in endothelial cell.
6-7. Growth of male gametocyte and formation of microgametes in endothelial cells of vessels.
  8. Fertilization of female gamete in an endothelial cell of blood-vessel.
  9. Oöcyst formed in endothelial cell.
10. Mature oöcyst containing sporozoites in endothelial cell. The escaping sporozoites enter red blood-corpuscles and are inoculated into tadpoles by the leech.

The infection of the frog is brought about by the leech, *Hemiclepsis marginata*, which introduces the sporozoites (Fig. 240). These make their way to and enter the endothelial cells of the capillaries of various organs of the body. Here each sporozoite becomes rounded, and commences to grow into a schizont, which eventually produces a large number of merozoites. The merozoites escape into the blood and infect other endothelial cells (Fig. 380, 1-4).

Nöller (1920*b*) found that merozoites of a special kind are finally produced, and that these, after entering endothelial cells, develop into micro- and macro-gametocytes. The microgametocyte produces a large number of microgametes, as in a typical *Eimeria*, and fertilization of the macrogamete ensues (Fig. 380, 6-8). An oöcyst is formed round the zygote, which breaks up directly, without the formation of sporoblasts and sporocysts, into a number of sporozoites (Fig. 380, 9-10). The latter, by rupture of the oöcyst, escape into the blood and enter red blood-corpuscles. Apparently the leech sucks up the sporozoites with the blood, and they enter the epithelial cells of the gut or wander about the tissues of the leech without further development. In some way not properly understood they gain entrance to another frog.

If this cycle of development is confirmed, it is a remarkable one in that the whole development, up to the formation of sporozoites, takes place in the endothelial cells of the blood-vessels, and is an illustration of a coccidium, originally transferred from host to host in the oöcyst stage as in the more typical forms, having become adapted to life in the blood-stream. In the majority of hæmogregarines, as will be seen below, it is the gametocytes which enter the circulating cells and are taken up by the invertebrate host, and not the sporozoites, as in this case, and it is in the invertebrate that fertilization and development of the oöcysts take place. In the case of *L. minima*, the life-cycle is like that of *Eimeria schubergi*, except that development occurs in the endothelial cells of the blood-vessels instead of those of the intestine. The possibility of the escape of the oöcysts to the exterior having been lost by this change of habit, the difficulty is overcome by the leech transferring the sporozoites from host to host. It will thus be seen that in the genus *Schellackia* schizogony and sporogony take place in the intestinal wall, as in a typical intestinal coccidium. In *Lankesterella* these stages of the cycle have been transferred to the endothelial cells of the vessels. According to Nöller, the stages of development of *L. minima* in the vessels have been mistaken for those of *Isospora lieberkühni* (see p. 828).

2. *Sub-Order:* **Hæmosporidiidea.**

This sub-order comprises the blood-parasites which are commonly referred to as hæmosporidia. As already remarked, their affinities are undoubtedly with the coccidia, from which they may be supposed to have evolved. This relationship will at once be realized if the life-cycles are compared (Figs. 337 and 391). In both cases the infection of the vertebrate is initiated by the entry of a sporozoite into a cell. The sporozoite becomes a schizont, which produces merozoites, and these again become schizonts, producing more merozoites. Eventually certain merozoites develop into male or female gametocytes. The former produces a number of male gametes, one of which fertilizes the female gamete. The zygote thus produced becomes enclosed in an oöcyst and divides into a number of sporozoites, which, by infecting the vertebrate, recommence the cycle. The hæmosporidia may be regarded as coccidia of the intestinal epithelium which have become adapted to a life in the circulating cells of the blood. In association with this change, certain differences arise. Whereas in the coccidia the fertilization process and the encystment of the zygote in a resistant oöcyst takes place in the vertebrate host, in the hæmosporidia the fertilization process takes place in the stomach of an invertebrate which has sucked up gametocytes from the blood of the vertebrate. The zygote, instead of being a motionless body, is a motile vermicule (oökinete) which penetrates the wall of the stomach and then forms its oöcyst, which is a delicate structure increasing in size with growth of the zygote. The latter eventually produces sporozoites, which escape into the tissues and are inoculated into the vertebrate when the invertebrate feeds on it. In the typical coccidia the male gametocyte produces male gametes after a relatively slow process of nuclear multiplication, while in the hæmosporidia the male gametes are formed by a violent process known as flagellation or exflagellation, which occurs in the stomach of the invertebrate. In the coccidia, again, the zygote is fully developed when fertilization has occurred, and when the tough oöcyst is formed no further increase in size takes place, whereas in the hæmosporidia the zygote forms a delicate oöcyst, which increases considerably in size after it has been formed. The oöcyst, in the case of coccidia, is a more durable structure, which persists after the sporozoites are developed, and protects them so long as they are exposed to the possibilities of desiccation outside the host. It is only dispensed with after it is taken into the intestine of the vertebrate. The oöcyst of the hæmosporidia only persists while the zygote is producing sporozoites. As soon as these are formed it ruptures, and the sporozoites escape into the tissues of the invertebrate, where they remain till they are injected into the vertebrate.

In the sub-order **Hæmosporidiidea** there are two families, the PLASMODIIDÆ and the HÆMOPROTEIDÆ. In the former, the whole of the vertebrate cycle of development occurs in the circulating red blood-corpuscles. The growth into schizonts of the sporozoites in the first place, and of the merozoites which are subsequently formed, takes place in these cells. Finally, certain merozoites, after entering red blood-corpuscles, instead of producing schizonts, develop into male and female gametocytes, which undergo no further development till they are taken up by an invertebrate host. It thus happens that the whole of the vertebrate cycle can be studied in the blood. In the Hæmoproteidæ, the only forms which occur in the circulating blood-cells are the gametocytes. The schizogony cycle occurs in another type of cell, which is probably one of the endothelial cells lining the blood-capillaries. In these cells, after several repetitions of schizogony, certain merozoites are produced which enter blood-corpuscles instead of the endothelial cells, and there develop into gametocytes which undergo no further change till the necessary environment of the invertebrate's intestine is reached. As the only forms occurring in the blood-cells are gametocytes, it follows that the whole cycle cannot be studied in the blood. The schizogony stages can be found only in smears or sections of the various organs in the vessels of which they occur.

It has been pointed out above (p. 878) that the sub-family Lankesterellinæ includes forms in which the whole cycle takes place in the endothelial cells of the blood-vessels, and that the sporozoites, when finally produced, enter the red blood-corpuscles to await transference to the invertebrate host. In this case, the production of gametocytes and the subsequent fertilization and formation of sporozoites occur in the endothelial cells. In the Hæmoproteidæ the evolution has progressed further, for the development up to the formation of the merozoites, which are in reality young gametocytes, occurs in the endothelial cells, but when the young gametocytes escape into the blood-stream, instead of entering endothelial cells again as in *Lankesterella minima* (Fig. 380), they penetrate the red blood-corpuscles, and no further development takes place there except a growth to their full size. The further development with fertilization and sporozoite formation takes place in the invertebrate. In the Plasmodiidæ another advance is made, for the sporozoites injected by the invertebrate, instead of entering the endothelial cells of the vessels to initiate the schizogony cycle, directly invade the red blood-corpuscles themselves, in which the whole schizogony cycle terminating in the production of gametocytes takes place.

A characteristic feature of the forms which penetrate the red blood-corpuscles and grow at their expense is the gradual deposition in their

PLATE VI.

HÆMOSPORIDIA OF BIRDS AS SEEN IN DRIED BLOOD-FILMS STAINED WITH ROMANOWSKY STAINS. (×2,000).

1-10. *Plasmodium præcox* from blood of Bagdad sparrow.
11-17. *Hæmoproteus oryzivoræ* from blood of Java sparrow (*Munia orizivora*).
18-20. *Leucocytozoon* sp. from a blood-film of a thrush (*Hylocichla musica*) which had been dead some time.
21-23. *Leucocytozoon neavei* from the blood of the guinea-fowl (*Numida ptilorhyncha*).

(ORIGINAL.)

PLATE VI

1 2 3 4 5

6 7 8 9 10

11 12 13 14 15

16 17 18 19 20

21 22

23
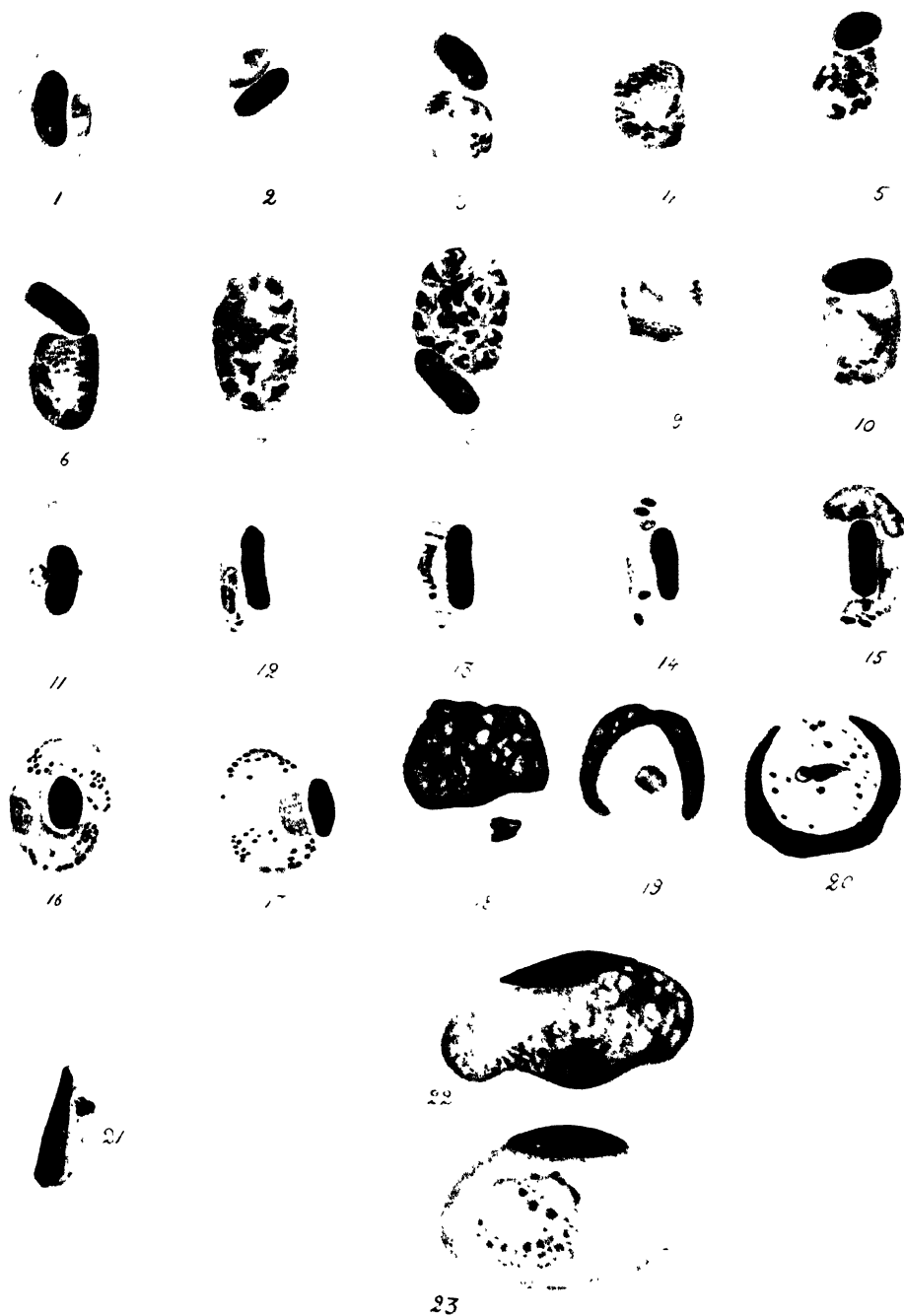
Bfotting

cytoplasm of granules of pigment derived from the hæmoglobin of the corpuscles. It is doubly refractile, of a brown or black colour, and is known as melanin or hæmozoin. It differs from hæmosiderin, a yellow pigment also derived from hæmoglobin and deposited in tissue cells, in that it does not give an iron reaction. It is soluble in solutions of ammonium sulphide and in acid alcohol.

1. *Family:* HÆMOPROTEIDÆ Doflein, 1916.

As indicated above, the members of this family are parasites of endothelial cells of the blood-vessels of vertebrates at one stage of their growth, of the circulating cells of the blood at another, and of the tissues of an invertebrate during sporogony (Fig. 383). The schizogony cycle occurs in the endothelial cells in which the young merozoite, and presumably the sporozoite when first injected, grow at the expense of the cells into large schizonts, which break up into a large number of merozoites. These invade other endothelial cells, and the process is repeated. Eventually, certain merozoites, instead of entering the endothelial cells, penetrate the circulating cells— either the red blood-corpuscles or others—where they grow into mature gametocytes, which undergo no further change till they find their way into their transmitting host, in which a development comparable with that of the human malarial parasites in the mosquito takes place. It is important to remember that in the peripheral blood of birds and reptiles, in which these parasites are found, only the gametocytes in various stages of growth occur, the schizogony cycle only being observed in sections or smears made from the internal organs.

There are two genera in the family Hæmoproteidæ. The first of these is the genus *Hæmoproteus* Kruse, 1890. It includes forms which reproduce asexually in the endothelial cells of the blood-vessels, while the young gametocytes enter the red blood-corpuscles (Fig. 381). The gametocyte, when fully formed, is halter-shaped, and is frequently called a halteridium (Plate VI., 11-17, p. 882). During its growth within the red blood-cell it produces pigment granules from the hæmoglobin. The second is the genus *Leucocytozoon* Danilewsky, 1890. It is probable that the asexual cycle is similar to that of *Hæmoproteus*, and occurs in the endothelial cells of the blood-vessels. The gametocytes, when formed, invade the circulating cells of the blood, but these are not normal red blood-corpuscles (Plate VI., 18-23, p. 882). Some have maintained that they are leucocytes, hence the name *Leucocytozoon;* but it seems more probable that they are immature red blood-cells which have not yet produced hæmoglobin. During growth of the gametocyte the cell is profoundly altered, and in most cases becomes an elongate spindle (Fig. 382). Furthermore, as no hæmoglobin occurs in the cell, the growing gametocyte does not form

pigment like the members of the genus *Hæmoproteus*. The complete development in the invertebrate host is known only in the case of the *Hæmoproteus* of the pigeon, its course being practically identical with that of the malarial parasite in mosquitoes (Fig. 383).
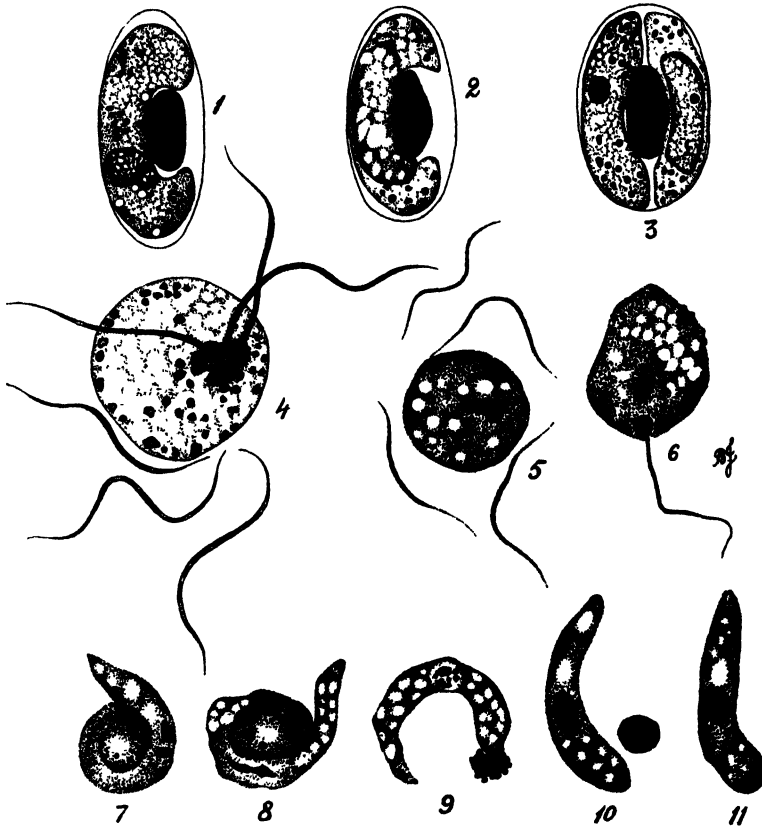
FIG. 381.—*Hæmoproteus* OF THE KESTREL (*Cerchneis tinnunculus*) (1-3, × 2,000; 4-11, × 1,800). (AFTER WASIELEWSKI AND WÜLKER, 1918).

1. Female gametocyte with compact nucleus.   2. Male gametocyte with large nucleus.
3. Male and female gametocyte—double infection.
4. Formation of microgametes—exflagellation.
5. Male gametes around female gamete.   6. Fertilization.
7-10. Oökinetes showing gradual separation of pigment.
11. Oökinete with pigment still present.

As gametocytes are incapable of reproducing in the vertebrate, it follows that infection cannot be conveyed directly from one vertebrate host to another by inoculation of blood. As will be shown below, with members of the genus *Plasmodium* this can be done, as the peripheral blood contains the asexually reproducing forms as well as the gametocytes.

Casagrandi and Barbagallo (1907) claimed to have infected chickens with the *Hæmoproteus* of the sparrow by blood inoculation. It seems evident that there must have been some fallacy in this observation.

Under natural conditions the gametocytes continue their development only in an invertebrate. The stomach phases of this development, including microgamete formation, fertilization, and production of the

FIG. 382.—*Leucocytozoon ziemanni* OF THE LITTLE OWL, *Athene noctua*, AS SEEN IN WET FIXED FILMS (× *ca.* 2,000). (1 AND 2 AFTER REICHENOW, 1920; 3 FROM RICHENOW, 1920, AFTER SCHAUDINN.)

    1. Macrogametocyte.   2. Microgametocyte.   3. Oökinete.

oökinete, can, however, be directly observed in freshly drawn blood between a slide and cover-glass, as first noted by MacCallum (1897) in the case of *Hæmoproteus* of birds (Fig. 381).

Genus: Hæmoproteus Kruse, 1890.

Members of this genus were first seen in birds by Danilewsky in 1889, since which date they have been recorded from some hundreds of different species of bird. The occurrence of another pigmented parasite in the blood of birds (*Plasmodium præcox*) naturally led to some confusion

between the two till they were properly differentiated. The fact that the adult gametocytes of members of the genus *Hæmoproteus* have the habit of encircling the nucleus of the red blood-corpuscles like a halter led Labbé (1894) to introduce the name Halteridium, by which these parasites are commonly known. The investigations of Danilewsky, Laveran, Kruse, Grassi, Feletti, and others indicated the difference between proteosoma (*Plasmodium*) and halteridium (*Hæmoproteus*), the two types of pigmented parasite found in the blood of birds. The researches of MacCallum (1897) explained the function of the puzzling " flagellating body," and demonstrated for the first time that this was a process by which the male gametocyte produced male gametes, which eventually fertilized female gametes and led to the formation of motile zygotes or oökinetes. Though the researches of Ross, referred to below, had elucidated the cycle of *P. præcox* of birds in *Culex fatigans*, observers failed to detect a similar development of halteridium in mosquitoes.

Schaudinn (1904), however, published a remarkable account of the development of *Hæmoproteus noctuæ*, the halteridium of the little owl, *Athene noctua*. He supposed that the intracorpuscular halteridium stages were merely developmental forms of a trypanosome. In the blood of the owl, the organism was supposed to be intracellular during the daytime, where it appeared as the well-known halteridium in various stages of growth, while at night it left the cell to become a trypanosome, which reproduced by division. Finally, when growth was complete, these migrations ceased, and the adult halteridium remained in the cell to continue its development in the mosquito, *Culex pipiens*. The process of microgamete formation and fertilization of the female gamete were described as taking place in the manner first noted by MacCallum, but the male gametes were stated to be of the nature of narrow trypanosomes. The oökinete, the further development of which had not been discovered, was described by Schaudinn as developing into a trypanosome after undergoing nuclear changes of a complicated nature. The trypanosomes formed from the oökinetes multiplied and populated the mosquito's stomach and intestine with flagellates, which were distinguished as male, female, and indifferent forms. Eventually it was claimed that some of these were inoculated to the owl by the mosquito, and the vertebrate cycle recommenced. Many careful attempts have been made to confirm Schaudinn's statements, but without success, so that the only possible conclusion is that his observations were the result of confusion of several distinct parasites. This is all the more certain in that the asexual cycle of certain members of the genus has since been discovered as taking place by schizogony in the endothelial cells, as first demonstrated by Aragão (1908), while Adie (1915) has elucidated the sporogony cycle of *H. columbæ* in *Lynchia maura*, which had

been proved to be capable of transmitting infection from pigeon to pigeon by Sergent, Ed. and Et. (1907).

Certain writers continue to support the views expressed by Schaudinn, and some go so far as to divide the genus *Hæmoproteus* into two groups, the members of one of which are supposed to follow Schaudinn's still unconfirmed cycle, while the members of the other develop along the line worked out by Aragão and Adie. The majority of observers, however, disregard Schaudinn's statements concerning the development of *H. noctuæ*, which were so much the result of theoretical bias as to make it completely unjustifiable to employ his figures and description, as has frequently been done, as the basis of what the life-cycle of trypanosomes should be.

A further generalisation, based on Schaudinn's views, has been made by Hartmann and others. It is concluded that not only halteridium, but all other pigmented blood-parasites, are closely related to the trypanosomes, which themselves are supposed to possess two nuclei. Granules, which sometimes occur in the cytoplasm of these parasites outside the nucleus, and which take a red colour with Romanowsky stain, have been homologized with the kinetoplast of trypanosomes. Woodcock (1909a) described such an extranuclear granule in the halteridium of the chaffinch, and, though he subsequently changed his opinion, considered he had obtained the first definite evidence in support of Schaudinn's views. As the writer (1910a) pointed out, it is not the discovery of such a granule which is of importance, but the conclusive evidence of its homology with the kinetoplast of trypanosomes. This evidence no one has yet produced, so that there is no justification for the inclusion of the trypanosomes and the pigmented parasites in a special order (Binucleata) of the Mastigophora. As noted above (p. 331), it has not yet been established that the kinetoplast of a trypanosome is in any way equivalent to a nucleus.

Simond (1901) described the first pigmented parasite from the blood of a cold-blooded animal, the tortoise *Trionyx indicus* of the Ganges. He named it *Hæmamœba metchnikowi*, but it unquestionably belongs to the genus *Hæmoproteus*. Since Simond's discovery was made, a number of similar forms have been described from cold-blooded animals. Nothing is known of these apart from the gametocytes which appear in the red blood-corpuscles, though in some instances male gamete formation has been observed to take place, as in the case of the bird parasites. Future investigations will probably show that they have a cycle of development similar to that of *H. columbæ*.

HÆMOPROTEUS OF BIRDS.

Members of the genus *Hæmoproteus* have been discovered in a large number of birds, but practically nothing is known of the possibility of one and the same species occurring in different hosts. The question is more difficult to investigate than in the case of *Plasmodium præcox*, where the susceptibility of birds can be tested by direct inoculation of the asexual forms in the blood. Many species of *Hæmoproteus* have been named, and, though certain differences in the gametocytes undoubtedly occur, it is clear that many of the species are not valid. In most cases, the name has been given on the assumption that each parasite is specific to the host in which it occurs. Though future investigations will probably prove that many species do actually exist, at the present time there are few data from which definite conclusions can be drawn. The writer has attempted without success to infect canaries by injecting sporozoites from the salivary glands of *Lynchia maura* infected from pigeons harbouring *H. columbæ*. Pigeons were readily infected in this manner.

One point in connection with the members of the genus *Hæmoproteus* should always be remembered. If blood containing them be observed between a slide and cover-glass, the mature gametocytes quickly contract to a spherical form in preparation for fertilization. The same change may occur in the blood-vessels of a bird after death, so that only the altered parasites in distorted cells will be found if blood-films are not made immediately death has occurred. The altered parasites bear a resemblance to the gametocytes of *P. præcox*, so that errors of diagnosis have frequently been made (Plate VI., 9-10, p. 882).

Hæmoproteus columbæ Celli and Sanfelice, 1891.—This halteridium is a parasite of the common pigeon, *Columba livia*, and has been recorded from South Europe, Africa, India, and South America. It will probably

1*a*-3*a*. Growth of female gametocyte in red blood-corpuscle.
1*b*-3*b*. Growth of male gametocyte.
4*a*-5*a*. Rounding off of female gametocyte and escape from cell.
4*b*-5*b*. Rounding off of male gametocyte and formation of male gametes.
   6. Fertilization.
7-12. Formation of oökinete, which finally makes its way through the stomach wall of *Lynchia maura*.
 13. Young oöcyst on stomach wall of *Lynchia maura*.
 14. Mature oöcyst filled with sporozoites, which eventually enter the salivary glands of the fly and are thence injected into the pigeon.
 15. Sporozoite entering a cell, probably endothelial cell, of a blood-vessel of the pigeon.
 16. Growth of the sporozoite in mononuclear cell.
 17. Primary segmentation into a number of uninucleate bodies (cytomeres).
18-21. Each uninucleate body increases in size and becomes multinucleate.
22-23. Segmentation into numerous minute young gametocytes which enter the red blood-corpuscles.
It is possible that the primary schizogony represented at 16 and 17 is repeated several times before the succeeding development occurs.

Fig. 383.—Diagrammatic Representation of Life-Cycle of *Hæmoproteus columbæ* (× ca. 1,200). (After Aragão, 1908, with Additions and Modifications after the Work of Adie, 1915, 1924.)

[*For description see opposite page.*

be found to have a much wider distribution, and to occur in other birds than the common pigeon.

As pointed out above, the forms which parasitize the red blood-corpuscles, and are encountered in ordinary blood-films, are the gameto-cytes. These occur in all stages of growth from the tiny rings, which result from the entry of a merozoite, to the elongate crescent or halter-shaped adult gametocytes which lie around the nucleus of the host cell in the typical halteridium manner (Fig. 383, 1-3).

Cycle in the Pigeon.—The asexual cycle of *H. columbæ* occurs in the endothelial cells of the blood-vessels of various organs of the body, par-ticularly the lungs, as first described by Aragão (1908), and subsequently by Negri (1913), Acton and Knowles (1914), and Gonder (1915). The writer has followed the development in English pigeons exposed to the bites of *Lynchia maura* which had fed on infected pigeons from Algeria, and in pigeons inoculated with sporozoites from the salivary glands of these flies. The actual behaviour of the sporozoites after their injection into the pigeon has not been followed, but it is presumed that they enter endo-thelial cells of the blood-vessels and grow into schizonts, which are such a conspicuous feature of the later stages of an infection. If the organs of pigeons are examined when young gametocytes are commencing to appear in the red blood-corpuscles, numerous schizonts in various stages of development are found, especially in the lungs. The youngest stages are minute cytoplasmic bodies with a single nucleus within the cytoplasm of an endothelial cell (Fig. 383, 15-16). According to Aragão, growth takes place, followed by nuclear multiplication, and finally segmentation into fifteen or more small unpigmented masses (cytomeres), each with a single nucleus (Fig. 383, 17). Each of these continues to grow, and its nucleus multiplies by repeated divisions till the cytoplasm of the cell, which has become considerably hypertrophied, is filled by a number of multinucleate bodies, each of which appears to be surrounded by a fine cyst wall or membrane. Within the membrane the multinucleate cyto-plasmic body or cytomere divides into an enormous number of minute merozoites (Fig. 383, 18-23). The development up to this stage occupies about four weeks, during which it is presumed that schizogony has been repeated a number of times. The endothelial cell finally breaks down, and the cysts, which vary much in size, but may reach a diameter of 60 microns, are set free and accumulate in the capillaries, which are some-times completely blocked (Fig. 389, 2). Shortly after their liberation, the cysts rupture and the merozoites escape into the blood-stream. It is then that the first forms are seen in the red blood-corpuscles as minute cyto-plasmic bodies with a single nucleus and often a vacuole (Fig. 383, 1). These have been formed by the entry of the merozoites liberated from the

cysts. It is possible that some of them enter other endothelial cells, and again become schizonts. The forms within the endothelial cells are never pigmented, since no hæmoglobin occurs in these cells. Acton and Knowles (1914), who studied the development of *H. columbæ* in the pigeon in India, came to the conclusion that schizogony occurred only in the lung, and that merozoites, when produced, either entered red blood-corpuscles, where they grew into gametocytes, owing to the blood offering unfavourable conditions, or remained in the lung, where they became schizonts in surroundings which were favourable owing to a greater supply of oxygen. In the sparrow of Bagdad the writer, however, noted that schizonts occurred in the vessels of the kidney and liver as also in those of the lung (Fig. 389). Other observers have also noted that in the case of *H. columbæ* schizogony is by no means limited to the lungs. Wasielewski and Wülker (1918), in the case of the halteridium of the kestrel (*Cerchneis tinnunculus*), noted that masses of schizonts occurred in the vessels of the lungs, liver, kidneys, and spleen. In the case of pigeons studied by the writer, the schizonts do not necessarily form cytomeres before giving rise to merozoites. The schizont is usually a large sausage-shaped body with innumerable nuclei. It is lodged in an endothelial cell of a capillary, and during its growth along the wall of the vessel it may send out branches along the bifurcations of the capillary, so that it becomes triradiate or even multiradiate.

The merozoites which have entered the red blood-corpuscles are young gametocytes. Sometimes as many as a dozen young forms are present in a single cell. In dry films, as pointed out by Wasielewski and Wülker (1918), by a fusion and overlapping of the parasites these multiple infections may give rise to an appearance of schizogony. When the gametocytes are fully grown, it is only rarely that even two are found in one cell, so that it would seem, as in the case of the malarial parasites of man, that either the cells with multiple infections die, or some of the parasites leave the cell. The young gametocyte very soon becomes elongate, and, as in members of the genus *Plasmodium*, granules of brown or black pigment appear in the cytoplasm (Fig. 383, 1-3). As increase in size takes place, the organism grows round one side of the nucleus, which is sometimes pushed to the side of the cell, which, however, retains its original shape and size. When fully formed, the elongate sausage-shaped gametocyte may completely encircle the nucleus. It contains a number of pigment granules distributed through its cytoplasm. The single nucleus is central in position. The adult gametocytes and even the younger ones can be distinguished as male and female. The former consists of hyaline cytoplasm, stains a pale blue or pinkish colour with Romanowsky stains, and has a rather large central nucleus consisting of a membrane enclosing a number of fine chromatin granules (Fig. 383, 1b-3b). The

female gametocyte has a denser cytoplasm, stains more deeply blue, and possesses a more compact nucleus, in which a distinct karyosome can be detected, especially when properly fixed (Fig. 383, 1a-3a). The pigment granules in the male gametocyte are frequently aggregated into a number of spherical masses, those in the female being more uniformly distributed through the cytoplasm. Occasionally, a cell will be seen with a gametocyte on each side of the nucleus, which retains its central position (Fig. 381 3). These may be of the same or of different sex.

Influenced by the fact that schizogony of proteosoma takes place in the red blood-corpuscles, Labbé (1894) describes what he supposed to be schizogony of halteridium in the peripheral blood. The forms which are now known to be gametocytes were supposed to break up into a number of merozoites at each end. Carpano (1913) seems to be the only observer who claims to have rediscovered this process, but as his so-called halter-shaped schizonts with merozoites at each end still have the central nucleus characteristic of the gametocyte, it seems probable that what he called nuclei at the poles were granules of another nature. The true schizogony is undoubtedly that which takes place in the endothelial cells. Attention has been drawn above to the occasional occurrence of one or more extra-nuclear red-staining granules in these gametocytes. Legroux and Lwoff (1924) have noted what they interpret as gametocytes of *H. oryzivoræ* with two, eight, and sixteen nuclei. They believe that these stages represent a parthenogenesis of the macrogametocyte. As noted above, Wasielewski and Wülker (1918) called attention to the fact that multiple infection of red cells might give rise to an appearance of schizogony owing to the actual fusion or the apparent fusion in dried films of the cytoplasm of a number of young parasites.

Cycle in the Fly.—The subsequent development of the gametocytes of *H. columbæ* takes place in *Lynchia maura* (Fig. 387). That this fly was the transmitting host was first proved by Sergent, Ed. and Et. (1906). A search for a developmental cycle in the fly revealed the male gametocytes producing male gametes by the active process of flagellation (Fig. 383, 4b-5b), the female gametocyte giving rise to a single female gamete by a process of maturation (Fig. 383, 4a-5a), and the fertilization of the female gamete by one of the male gametes (Fig. 383, 6). The zygote thus formed was seen to become an oökinete or travelling vermicule (Fig. 383, 7-12).

The development up to this stage is comparable with that of the parasites of human malaria to be described below. It can readily be observed in fresh blood between a slide and cover-glass. The further development of the oökinetes could not be traced, though it was noted that they got rid of their pigment, as was subsequently observed again by Mezincescu (1909) and Gonder (1915). Woodcock (1915) noted that

the oökinetes of the parasite of the little owl were devoid of pigment in
the stomach of *Culex pipiens*, while Wasielewski and Wülker (1918)
described the separation of the pigment from the oökinetes of the parasite
of the kestrel (Fig. 381).   The pigment accumulates at the posterior end,
and is separated with a small portion of the cytoplasm by a constriction.
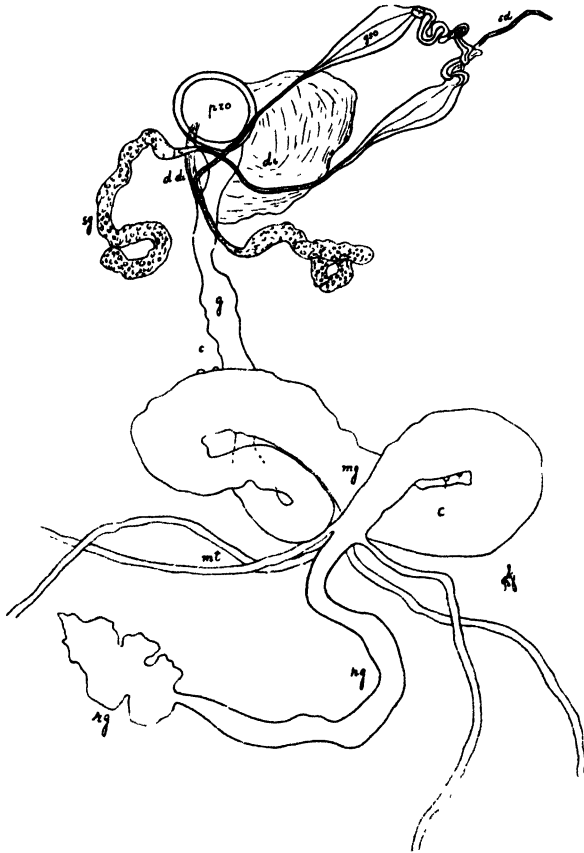
FIG. 384.- DISSECTION OF THE ALIMENTARY TRACT AND SALIVARY GLANDS OF
   Lynchia maura, SHOWING OÖCYSTS OF *Hæmoproteus* OF THE PIGEON ON THE
   WALL OF THE MID-GUT.   (AFTER HELEN ADIE, 1915.)

s.d., Common salivary duct; *g.s.o.*, goblet-shaped organ in thorax; *pro.*, proventriculus; *di* . pro-
   ventricular diverticulum; *d.di.*, duct of diverticulum; *s.g.*, salivary gland; *m.g* , mid-gut;
   c., oöcysts on surface of gut; *m.t* , Malpighian tubes; *h.g.*, hind gut; *r.g.*, rectal glands.

If all the pigment be not removed by the first, a second constriction forms.
In view of the observation of Adie, to be described below, it would seem
that the separation of the pigment may be an abnormal process.

   Neither the Sergents (1906), Aragão (1907, 1908), nor Gonder (1915)
were able to trace any further development of the oökinete in *L. maura*,

and it was concluded that in all probability the oökinete itself was inoculated into the pigeon. In fact, Mezincescu (1909) and Gonder (1915) claim to have infected pigeons by injecting oökinetes. Aragão (1916a), however, attempted to repeat this experiment without success. Helen Adie (1915), working in India, was the first to follow the complete development of the oökinetes in *L. maura* (Fig. 383, 13-14). They penetrate the hinder portion of the mid-gut, where they produce pigmented oöcysts
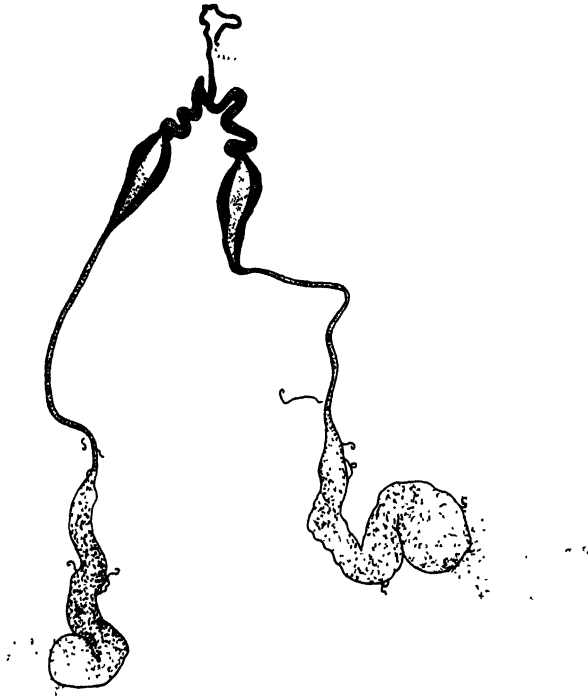
FIG. 385.—DISSECTION OF THE SALIVARY APPARATUS OF A *Lynchia maura* HEAVILY INFECTED WITH SPOROZOITES OF *Hæmoproteus* OF THE PIGEON. (AFTER HELEN ADIE, 1915.)

The granular material around the cut end of the common salivary duct and the extremities of the salivary glands represent escaping sporozoites, the shape and structure of which are not apparent at this low magnification.

on the outer surface of its wall, as malarial parasites do in mosquitoes (Fig. 384). These oöcysts increase in size, and eventually give rise to numbers of sporozoites, which are of the type seen in malarial parasites, and measure up to 10 microns in length. They invade the salivary glands which lie ventrally in the abdomen, and which in infected flies often appear packed with sporozoites (Fig. 385). The minute details of the development were not given, but it was said to follow very closely that of *Plasmodium præcox* in species of *Culex*. The oöcysts, which moreover contain

pigment, are not so readily detected on the gut of *L. maura* as they are in
mosquitoes, on account of the size of the gut itself.   If, however, the
mid-gut is under a cover-glass, the cysts can be seen at the edge, and by
moving the cover-glass so that the gut rolls on the slide, successive crops
of oöcysts will come into view.   In a later paper Adie (1924) describes
observations made on pigeons in Sergent's laboratory in Algiers.   She
has been able to confirm completely her previous work in India, and gives
figures of the developmental stages of the parasite (Fig. 386).   Flies which
have lived on infected pigeons for ten to twelve days, since they feed
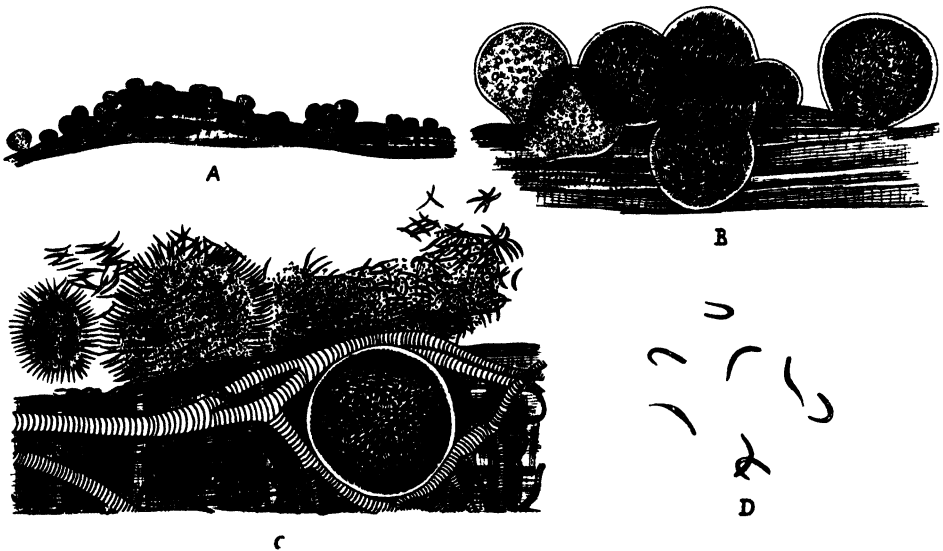
FIG. 386.-- DEVELOPMENT OF *Hæmoproteus columbæ* ON STOMACH OF *Lynchia maura*.
(AFTER HELEN ADIE, 1924; SLIGHTLY ALTERED.)

A. Side view of edge of flattened stomach, showing numerous oöcysts of various sizes (× *ca*. 72).
B. Edge of stomach more highly magnified, showing mature and immature oöcysts with
   pigment granules (× *ca*. 450).
C. Intact oöcyst and ruptured oöcyst, with discharged sporozoites and residual cytoplasm
   (× *ca*. 600).   D. Free sporozoites (× *ca*. 1,200).

daily, show all stages of development of the parasite from the oökinete up
to the sporozoites in the salivary glands.   The mature oöcyst measures
about 36 microns in diameter.   Clean pigeons exposed to infection by
infected flies first show young gametocytes in the blood in about four weeks,
and a similar interval is noted when sporozoites from dissected flies are
injected subcutaneously.   The writer has succeeded in infecting pigeons in
England by means of flies sent from Algiers.   With the help of Mrs. Adie he
has also been able to demonstrate the various stages of the development
in the fly, so that there is no longer any doubt that the cycle first described

by her in 1915 is the correct one. The various stages of development correspond very closely with those of malarial parasites in mosquitoes.

Transmission.—As already noted, Sergent, Ed. and Et. (1906), first actually transmitted the infection experimentally to pigeons by means of infected flies (*Lynchia maura*) which were sent from Algiers to Paris (Fig. 387). In South America, according to Aragão (1916a), the transmitting hosts of *H. columbæ* are *L. lividicolor, L. brunea*, and *Microlynchia fusilla*. Working in South Africa, Gonder (1915) effected transmission with *L. (Olfersia) capensis*, and Adie (1915, 1924) in India and Algeria with *L. maura*. The writer has infected English pigeons in London by means of this fly fed on Algerian birds.
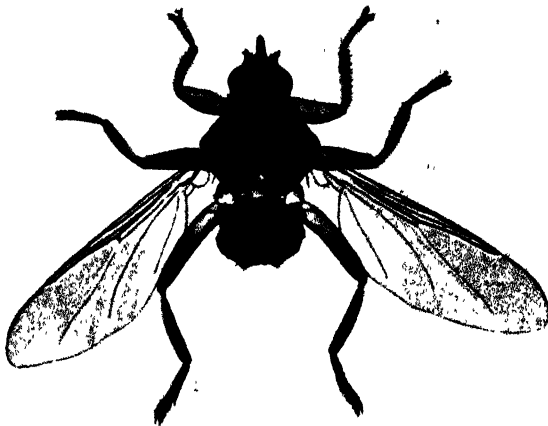
Fig. 387.—*Lynchia maura* (♀), THE TRANSMITTING HOST OF *Hæmoproteus columbæ* (× 5). (ORIGINAL.)

OTHER OBSERVATIONS ON HALTERIDIA OF BIRDS.—Mello and Braz de Sa (1916) described the schizogony cycle of a halteridium of an Indian pigeon (*Copsychus saularis*), Aragão (1916a) that of the halteridia of *Columbigallina talpacota* and *Chanthornus jamaicai* of Brazil, and Wasielewski and Wülker (1918) that of the halteridium of the falcon (*Cerchneis tinnunculus*). A similar cycle was demonstrated for *H. oryzivoræ* of the Java sparrow (*Munia oryzivora*) by Anschütz (1909, 1910), who observed the schizonts, not only in the lungs, but also in the bone marrow and brain (Plate VI., 11-17, p. 882). The writer found schizonts in the lung, liver, and kidney of the common Bagdad sparrow (Figs. 388, 389).

Franchini (1922a) studied the development of *H. chelidonis* of the Italian swallow (*Chelidon urbica*). He has seen developmental forms in

the lungs of the type described above, and he also claims to have seen very similar stages in the tissues of fleas (*Ceratophyllus hirundinis*) taken from abandoned nests. He supposes the forms seen in the fleas to be the stages of development in the invertebrate host.

Aragão observed that oökinetes of *H. columbæ* were formed in the intestine of the mite (*Dermanyssus*) and in the mosquitoes (*Culex tæniorhynchus*, *C. confirmatus*, and *Cellia argyrotarsus*). Nöller (1920*a*) noted that oökinetes could be developed in the stomach of the bed bug. He found no development in mites (*Dermanyssus*) nor in fleas (*Hectopsylla psittaci*), but in *Aëdes argenteus* (*Stegomyia fasciata*) oökinetes formed quite readily, and at a temperature of 11° to 12° C. persisted for at least six days. Wasielewski

and Wülker (1918) point out that in kestrels the infection is either acute or chronic. The acute one occurs in young birds, which show very heavy infections with schizonts in the various organs. This phase subsides and is succeeded by a chronic one characterized by relapses of a milder type, which occur during the course of several years. As Danilewsky (1888) and Koch (1899) had done before, they noted that young birds in the nest were most heavily infected, and believe that the apterous fly (*Carnus hemapterus*), which is found on young nestlings, will probably prove to be the transmitting host of the parasite of the kestrel.

FIG. 388. — *Hæmoproteus* OF BAGDAD SPARROW MEROZOITES IN VACUOLE IN MONONUCLEAR CELL IN SECTION OF LIVER (× 2,000). (ORIGINAL.)

These presumably become schizonts which give rise to similar merozoites again, or become the large schizonts which produce young gametocytes as in Fig. 389, 1 and 2.

Sergent and Béguet (1914) showed that pigeons which had recovered from an infection of *H. columbæ* were not immune to reinfection. In the case of the sparrow, *Passer chloris*, Sergent, Ed. and Et. (1907), showed that, without reinfection, the parasites might persist in the blood for three years. They demonstrated a similar persistence of infection in the pigeon. Gametocytes might be absent from the blood for months together, but would reappear later. This observation has been repeated by Senevet and Witas (1922), who point out that on this account it is exceedingly difficult to decide whether a bird is or is not infected.

HÆMOPROTEUS OF COLD-BLOODED ANIMALS.

As already stated, the first pigmented parasite of a cold-blooded animal to be described was one which Simond (1901) discovered in the Indian river tortoise, *Trionyx indicus*. He gave it the name *Hæmamœba*
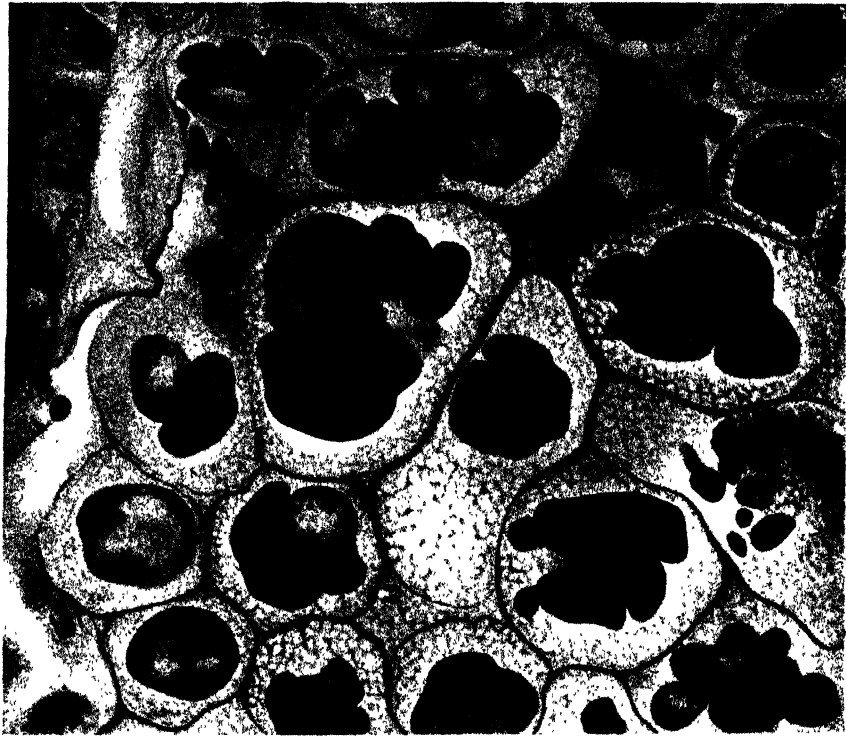
FIG. 389.—*Hæmoproteus* OF BAGDAD SPARROW: SCHIZOGONY (FORMING GAMETO-
CYTES) AS SEEN IN SECTIONS OF TISSUES FIXED IN ZENKER'S FLUID (× 1,000).
(ORIGINAL).

For description see opposite page.

metchnikowi.   Laveran (1905) described as *H. testudinis* a form seen by him in the South African tortoise *Testudo pardalis*, Bouet (1909) recorded as *Plasmodium roumei* a parasite of the West African tortoise, *Cinixys belliana*, and Johnston and Cleland (1909) a similar one, which was named *Hæmocystidium chelodinæ*, from the Australian tortoise, *Chelodina longicollis.*   In the following year the last-named observers found the same parasite in *Emydura krefftii* and *C. oblonga.*   All these parasites belong to the genus *Hæmoproteus.*   Pittaluga (1912) described *Hæmoproteus cajali* of *Clemmys africana* of New Guinea, and in the same year Plimmer recorded his discovery of a *Hæmocystidium* in five chelonians—viz., *Staurotypus triporcatus* of British Honduras, *Chrysemys picta* of North America, *Cinixys homeana*, *C. erosa*, and *C. belliana* of West Africa.   Joyeux (1913), again, saw the parasite in *C. belliana.*   The pigmented parasites of chelonians are thus of wide distribution.   It is very probable they all belong to the one species, *Hæmoproteus metchnikowi.*   Castellani and Willey (1904), who founded the genus *Hæmocystidium*, described as *H. simondi* a parasite of the Ceylon gecko, *Hemidactylus leschenaulti.*   This organism was studied later by Robertson (1908) and Dobell (1910a), and what is probably the same species was seen by de Mello (1916) in *H. brookei* in India and named by him *Hæmocystidium kopki.*   Dobell described schizogony as occurring in the red blood-corpuscles, but the appearance of the parasite suggests a *Hæmoproteus* rather than a *Plasmodium*, and it seems not improbable that the schizonts described by Dobell are in reality the result of multiple infection of red cells by young gametocytes.   *Hæmoproteus agamæ*, described by the writer (1909) from the lizard, *Agama colonorum*, of the Sudan, belongs to the genus *Plasmodium.*

Shortt (1922) gave the name *Hæmoproteus phyllodactyli* to a form discovered in the Persian gecko, *Phyllodactylus elisæ*, and the name *H. grahami* to another in the Persian rock lizard, *Agama nupta.*   These are very similar to and possibly identical with *H. simondi.*   Another form which may belong to the genus *Hæmoproteus* was discovered by Iturbe and Gonzalez (1921) in a lizard, *Anolis biporcatus*, in Venezuela.   It was named *Plasmodium gonzalezi*, though the only forms seen appeared to be gametocytes.

Pigmented parasites of snakes belonging to the genus *Hæmoproteus* have also been observed.   The first of these to be seen was one described by Bouet (1909) from a species of *Naja* or *Sepedon* of West Africa.   He

1. Numerous schizonts in blood-vessel of liver, each with several nuclei.
2. View of portion of a blood-vessel in the kidney blocked with mature schizonts ready for segmentation into young gametocytes.
3. Products of schizogony—young gametocytes scattered in the liver tissue.
4. Young gametocytes just after invasion of red blood-corpuscle.
5. Partially grown gametocytes in red blood-corpuscle.
6. Two gametocytes still further developed.   7. Mature gametocyte.

named the organism *Plasmodium mesnili* (Plate XVII., 1-5, p. 982). In the same year the writer (1909) described the same organism under the name *Hæmocystidium najæ* from *Naja hajæ* and *N. nigricollis* of the Sudan, while Leger, A. and M. (1914), observed it in *Sepedon hæmocholis* of Senegal. A pigmented parasite of an unnamed lizard was seen by Minchin (1910) in Uganda.

As regards the development of these forms, nothing is known with the exception of the fact that Joyeux (1913) noted that *Hæmoproteus roumei* behaved *in vitro* like the halteridium of birds in that the male gametocytes flagellated and produced male gametes. There is no information regarding the asexual reproduction and the invertebrate hosts, though in the case of the aquatic tortoises, leeches; and in the case of the snakes and lizards, ticks or mites, may be responsible for transmission.

The systematic position of these pigmented parasites was discussed by the writer (1915). It was concluded that they belonged either to the genus *Plasmodium* with schizonts in the red blood-corpuscles, or to the genus *Hæmoproteus* when no such forms occur in those cells. In both genera gametocytes are formed in the red blood-corpuscles. Shortt (1922) expressed a similar opinion. Many of these parasites have the same appearance as halteridium of birds, and the gametocytes can be distinguished as male and female. Young gametocytes in various stages of growth occur. Furthermore, flagellation in the halteridium manner also takes place, so that there seems no reason to include these forms in a separate genus, *Hæmocystidium*, since they correspond in every way with members of the genus *Hæmoproteus,* in which they will be included here. The forms which have been described from tortoises under various names are very similar to one another, so that it is not improbable that they belong to one species (*H. metchnikowi*).

Hæmoproteus roumei (Bouet, 1909).—In this parasite male and female gametocytes alone are known. The male forms are either spherical, with a diameter of 9 microns, or definitely elongate, and measuring 14·4 by 3·6 microns. The females are large, and are 12·6 microns in diameter when spherical, and 12·6 to 16·2 by 10·8 microns when elongate. The red cells, which normally measure 19·8 by 11 microns, may be increased in size. There is no distortion of the cell, though its nucleus may be displaced. *H. roumei* occurs in the tortoise, *Cinixys belliana,* of West Africa.

Hæmoproteus testudinus (Laveran, 1905).—Male and female gametocytes can be distinguished by differences in staining reaction. They are either ovoid or reniform, with a measurement of 10 to 12 microns in longest diameter, when they occupy one end of the cell, or they are elongate and measure 20 by 7 or 8 microns, and lie around the nucleus in the halteridium manner. The youngest gametocytes seen had a diameter of about

3 microns. The red cell, which has a length of 20 microns, is not distorted. The host is *Testudo pardalis* of South Africa.

Hæmoproteus chelodinæ (Johnston and Cleland, 1909).—This parasite occupies the end of the red cells, and varies in size from 4 by 3 to 12·5 by 10 microns. The normal measurements of the red cells, which are not distorted, are 17 to 18·5 by 10 to 12 microns. Male and female gametocytes can be distinguished. The parasite occurs in various Australian tortoises (*Chelodina longicollis, C. oblonga, Emydura krefftii*).

Hæmoproteus metchnikowi (Simond, 1901).—The only forms seen were gametocytes, which occupied one end of the red cell and had a diameter of 6 to 10 microns. The red cells, which have a length of about 20 microns, were not distorted. In some cases, a blunt prolongation of the parasite extended up the side of the nucleus. In no case were typical halteridium forms seen. Faintly staining male gametocytes with coarse irregularly distributed pigment and deeply-staining female gametocytes with finer pigment grains could be distinguished. The parasite was discovered by Simond (1901) in the Indian river tortoise, *Trionyx indicus*.

Hæmoproteus cajali Pittaluga, 1912.—This form resembles *H. metchnikowi*, except that some of the gametocytes extended round the nucleus in the halteridium manner. Young gametocytes were also seen. It was described by Pittaluga from *Clemmys africana* of New Guinea.

Hæmoproteus mesnili (Bouet, 1909).—This is a parasite of certain African cobras, and was first seen by the writer in 1907, though not described till 1909, after Bouet's account had appeared. The only forms seen are mature or immature gametocytes (Plate XVII., 1-5, p. 982). The smallest forms are non-pigmented bodies 2 to 3 microns in diameter. As they increase in size pigment grains appear, and growth round the nucleus of the red cell takes place, as in halteridium. The mature gametocytes may be as much as 21 microns in length. The normal red cell measures about 17 by 8·5 microns, and this may be increased to 25 by 14 microns in the case of infected cells. The cell is not distorted, nor is its nucleus displaced unless two parasites occur in the cell. The mature gametocytes and even the younger forms can be distinguished as male and female.

In the case of *Naja hajæ* and *N. nigricollis*, studied by the writer, very heavy infections occurred, many parasites being present in each field of the microscope. The appearance produced by these intense infections with the large nucleated red blood-corpuscles containing the highly pigmented gametocytes in all stages of growth was a very striking one. Sections of the organs, though showing the smaller vessels packed with parasites, did not reveal any schizogony stages like those known to occur in the case of the *Hæmoproteus* of birds. In order to discover these, if they occur, it would be necessary to examine the snakes at the early stages of an

infection. *H. mesnili* is a parasite of the African cobras, *Naja hajæ*, *N. nigricollis*, and *Sepedon hæmocholis*.

Hæmoproteus simondi (Castellani and Willey, 1904).—This organism occurs in the tree-dwelling gecko, *Hemidactylus leschenaulti*, of Ceylon. Its discoverers gave it the name *Hæmocystidium simondi*, but, as pointed out above, there is little justification for the recognition of a separate genus. The parasite was studied also by Robertson (1908) and Dobell (1910*a*). The gametocytes, both male and female, are large and completely fill the cells, which are distorted and have their nuclei displaced. The length of the gametocytes is about 18 microns, and the breadth about half this. There are numerous dark pigment granules distributed through the cytoplasm. Dobell describes schizonts in the red blood-corpuscles. These are round bodies about 8 microns in diameter, and are supposed to divide into two or four merozoites. It has still to be demonstrated that the so-called schizonts are not the result of multiple infections of a single cell with fusion of the cytoplasm of adjacent parasites. Wasielewski and Wülker (1918), as noted above, have shown that a similar appearance occurs in the case of the halteridium of the kestrel, and have demonstrated that it results from the fusion of adjacent parasites during the drying of the blood-film. What is probably the same form was described by de Mello (1916) under the name of *Hæmocystidium kopki* from *Hemidactylus brookei* of India, while Adler informs the writer that he has seen it in *H. turcicus* in Palestine.

Hæmoproteus phyllodactyli Shortt, 1922, and **H. grahami** Shortt, 1922. —These two parasites were discovered by Shortt in two Persian lizards, *Phyllodactylus elisæ* and *Agama nupta*. They closely resemble one another, and also *H. simondi*. The appearance of the mature gametocytes and the younger forms of these is very like that of the halteridia of birds. Schizonts were not observed, though these were searched for in smears and sections of the organs. Shortt rightly placed these parasites in the genus *Hæmoproteus*.

Hæmoproteus gonzalezi (Iturbe and Gonzalez, 1921).—This parasite was discovered by Iturbe and Gonzalez (1921) in a chameleon (*Anolis biporcatus*) of Venezuela. It was only seen on one occasion, the lizard having a heavy infection of what appeared to be gametocytes only. These occupied the sides of the red blood-corpuscles, and each was an ovoid body with a central or terminal nucleus. The pigment was in the form of coarse granules. No satisfactory evidence of schizogony in the peripheral blood was obtained, and it is suggested that it may take place in the internal organs. The parasite was placed in the genus *Plasmodium*, but on account of the absence of information regarding the schizonts, it is transferred to the genus *Hæmoproteus*.

Genus: Leucocytozoon Danilewsky, 1890.

The name *Leucocytozoon* was first given to certain unpigmented parasites seen by Danilewsky in the blood of birds. He suggested this name because he considered them to be parasitic within leucocytes, and though this may not actually be the case, the name still stands. The members of the genus are parasitic in certain cells of the blood, which contain no hæmoglobin, and in consequence they do not form pigment, like the nearly related members of the genus *Hæmoproteus*. The forms found in the peripheral blood are all gametocytes, which may be seen there in various stages of growth (Plate VI., 18-23, p. 882). As in the case of halteridium, they can be distinguished as male and female, the former staining more lightly than the latter, which consists of denser cytoplasm. The nucleus of the male is large, and contains within its membrane numerous chromatin granules, while that of the female is more compact and has a well-marked karyosome. The cells in which the youngest forms occur appear to be immature red blood-corpuscles devoid of hæmoglobin (Fig. 390, 1-2). As the gametocyte grows, the cell is profoundly changed and generally develops into an elongate fusiform body much larger than the normal red blood-corpuscles (Fig. 390, 3-5). The central part of this body is occupied by the elongate and hypertrophied nucleus of the cell and the elongate gametocyte. When observed in fresh blood preparations, as was first noted by the writer (1909) in the case of *L. neavei* of the guinea-fowl (*Numida ptilorhyncha*), the parasites are in constant activity. Peristaltic waves of contraction pass along the parasite first in one direction, then in the other, causing the cytoplasm of the parasite to be driven into the spindle-like extremities of the host cell. It was suggested that the peculiar shape of the cell might be accounted for by the constant pressure exerted in the longitudinal direction. During the activities of the parasite, the tapering extremities of the cell may show certain movements, but these are purely passive. As in the case of *Hæmoproteus*, the mature gametocytes may sometimes proceed to further development between slide and cover-glass. They leave the host cells, and the females become rounded macrogametes, while the males produce microgametes by a process of flagellation. Eventually, as was first observed by Danilewsky (1889), a large unpigmented oökinete is formed (Fig. 382). Furthermore, the observations of Moldovan (1914) on *L. ziemanni* indicate that the asexual reproduction takes place in the internal organs of the bird, probably in the endothelial cells of the vessels, as in the case of *Hæmoproteus*. It seems clear that the genus *Leucocytozoon* must find a place near the genus *Hæmoproteus*, from which it differs chiefly in the absence of pigment in the gametocytes and the peculiar effect it has on the host cell. There seems

to be little argument in favour of including the leucocytozoa with the coccidia or considering them as nearly related to the hæmogregarines, as Reichenow (1912) and Doflein (1916) have done.

As regards Schaudinn's (1904) remarkable views as to the affinities of these parasites little need be said. He mistook entirely the structure of the parasite *L. ziemanni* as it occurs in the little owl, *Athene noctua*, and considered the host cell as part of the parasite. The parasite itself was regarded as the endoplasm, and the spindle-like prolongations of the host cell cytoplasm as the ectoplasm. The nucleus of the host cell was explained as the nucleus of a leucocyte which the fusiform parasite had absorbed. These views were the outcome of a desire to homologize the parasite with a trypanosome, the only real resemblance to which was its elongate fusiform shape. Furthermore, Schaudinn described a complicated development in *Culex pipiens*, in which the oökinete was supposed to give rise to an enormous number of spirochætes, which themselves were considered to be very narrow trypanosomes. The whole of this work, as also that on *Hæmoproteus noctuæ* noted above, has received no confirmation, and cannot be accepted as correct.

A large number of leucocytozoa has been described from birds, and many of them have received specific names. It is exceedingly doubtful if any of these can be identified apart from their hosts. There is a great uniformity in the group as far as it is known. In describing these parasites, many observers have neglected the fact that they are large and easily deformed in the usual process of blood-film work. Working with *L. neavei*, the writer noted that, whereas in fresh blood preparations all the parasites were in spindle-shaped cells, in dried films, especially if made some time after the death of the bird, there was a much greater diversity of shape, many of the parasites being spherical, while the cells appeared to have lost their tail-like prolongations. The change is often the result of a rounding-off of the parasite in preparation for the fertilization process. As in the case of *Hæmoproteus*, this will occur in the blood-vessels of a bird after it has been killed. It is undoubtedly a fact that many of the descriptions and figures given of species of this genus have been made from forms which have altered in this manner. In some cases, however, it may be that the host cell is actually rounded, and does not possess the filamentous prolongations (Plate VI., 18-20, p. 882).

1-11. *Leucocytozoon neavei* from blood of guinea-fowl (× 2,000).
1-2. Hæmoglobin-free cells from blood; the types which appear to be those infected.
3. Very much flattened cell with young parasite.
4-7. Partially grown gametocytes in spindle-shaped cells.
8. Double infection of cell with partially grown forms.   9. Male gametocyte.
10. Female gametocyte.   11. Male and female gametocyte in same cell.
12-14. Schizogony of *L. ziemanni* in organs of little owl (× 1,600).
15-17. Diagram of supposed schizogony of *L. lovati* in organs of grouse (× 1,600).
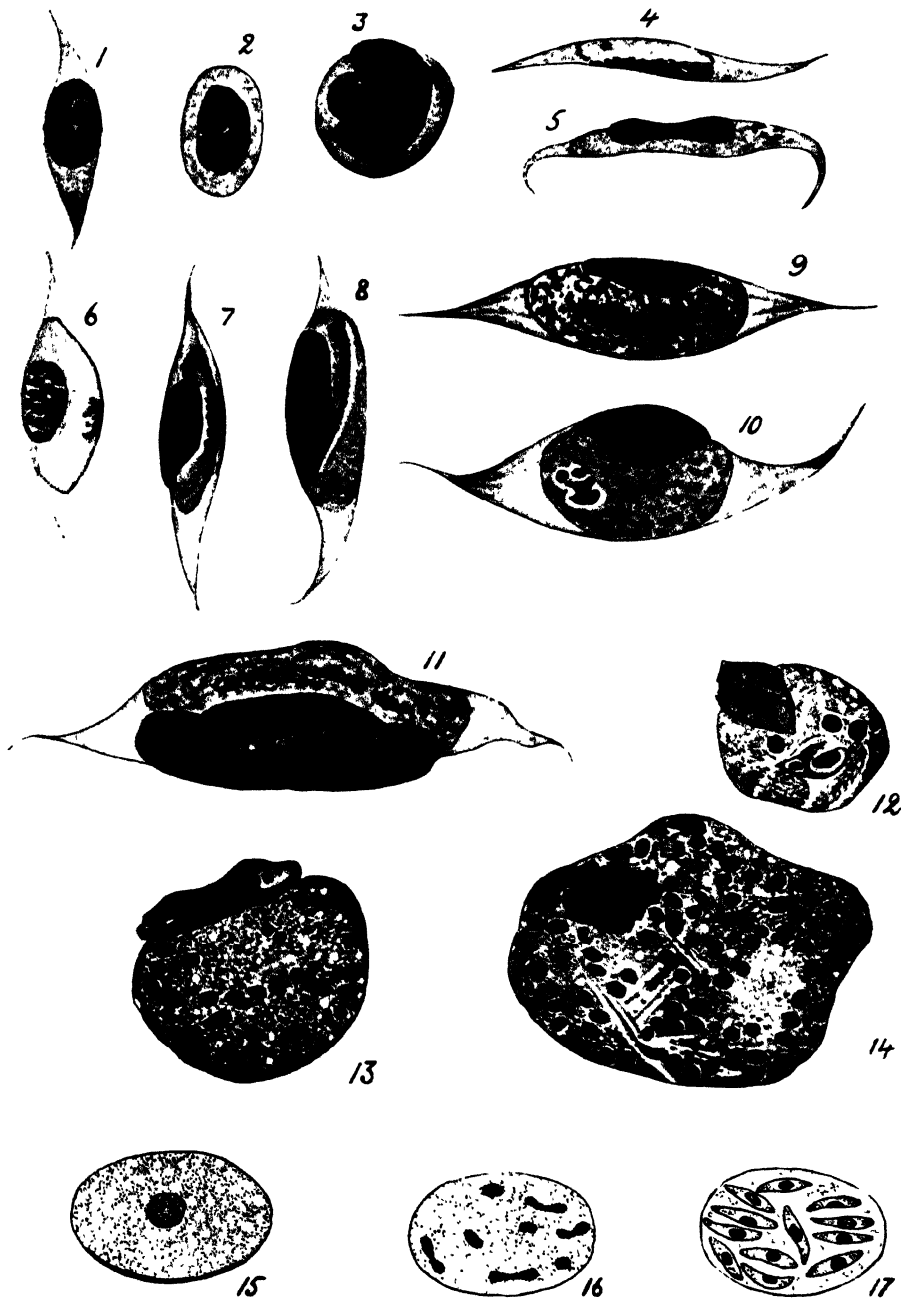
FIG. 390.—VARIOUS SPECIES OF *Leucocytozoon* FROM THE BLOOD AND ORGANS OF BIRDS. (1-11 AFTER WENYON, 1909; 12-14 AFTER MOLDOVAN, 1914; 15-17 AFTER FANTHAM, 1910.)

For description see opposite page.

Leucocytozoon neavei (Balfour, 1906).— This parasite was discovered by Neave (1906) in the Sudan guinea-fowl, *Numida ptylorhyncha,* and named *Hæmamœba neavei.* It was studied by the writer (1909). The youngest forms seen in the peripheral blood are minute cytoplasmic bodies about 3 microns in diameter, lying in the hollow of a nucleus in a more or less rounded cell. Slightly larger and elongate forms are also seen in cells which have a spindle shape (Fig. 390, 3-5). It is a difficult matter to determine the exact nature of these cells. In the blood of birds there occur cells which resemble the red blood-corpuscles, but appear to be devoid of hæmoglobin. Sometimes these cells are drawn out at their extremities, just as occurs in cells infected with the parasite (Fig. 390, 1-2). A comparison of these uninfected spindle cells with the smallest infected cells of the same shape lends support to the view that it is this type of cell which is invaded by the parasite. The cells are generally regarded as immature red blood-corpuscles. Whatever may be their nature, with increase in size of the parasites, which, it must be remembered, are gametocytes, the host cells grow considerably in size, and at the same time retain the spindle shape. At a very early stage, and during subsequent development, the gametocytes can be differentiated into male and female. The former consists of more hyaline cytoplasm, stains a pale blue or pink colour with Romanowsky stains, and has a nucleus represented by an elongate mass of granules. The female, on the other hand, consists of denser cytoplasm, which stains a deep blue colour, and has a compact nucleus consisting of a group of fine granules, and frequently one larger granule, which probably represents the karyosome. This is borne out by the appearances seen in specimens stained after wet fixation (Fig. 382). When fully grown, the gametocyte measures about 20 to 25 microns in length by about 5 microns in breadth. The nucleus of the host cell lies at one side of the parasite, and may be as long and as broad as the parasite itself. The central part of the cell is completely filled by the parasite and the nucleus, but terminally its cytoplasm is continued into tapering extremities 10 to 15 microns in length. The entire structure, made up of host cell and parasite, may measure 50 microns in length by 5 to 10 microns in breadth. As a rule, there is only a single parasite in each cell, but occasionally two gametocytes may be present, in which case the nucleus of the cell usually lies between them (Fig. 390, 8-11). In these cases the gametocytes may be of like or different sex. This is exactly comparable with what occurs occasionally in species of *Hæmoproteus* (Fig. 381, 3). The movements of the gametocytes, as seen in fresh blood, have been mentioned above, and they are to be compared with those of halteridium or the gametocytes of malarial parasites which occur prior to gamete formation. Though the process of flagellation or microgamete formation was not actually observed in *L. neavei,* an

appearance which must be interpreted as the fertilization of the female gamete by male gametes was seen in a stained film. As pointed out above, Danilewsky (1889-1891) observed the whole process up to the formation of the oökinete in the case of leucocytozoa of European birds. The male gametocyte, when producing male gametes (the flagellating body), he called the *Polymitus major*, and the oökinete the *Pseudovermiculi sanguinis.*

The degree to which guinea-fowls are found naturally infected varies considerably. The heaviest infections were seen in young birds, while older birds had, as a rule, scanty infections. This suggests that the young birds are infected soon after hatching. No asexual reproduction phases were observed in guinea-fowls, though these were specially looked for. It is probable that they would only be found in early stages of infection, as occurs in halteridium. Accounts of the asexual reproduction have been given for other species. Fantham (1910) described what he considered to be the schizogony of *L. lovati* of the British grouse, *Lagopus scoticus.* Ovoid encysted bodies measuring 11 to 14 by 8 to 11 microns were noted in the spleen. In the early stages there was a single nucleus, but older forms possessed twelve to twenty nuclei (Fig. 390, 15-17). Segmentation into a corresponding number of merozoites then occurs. These measure 7 to 8 microns in length by 1 to 1·5 microns in breadth. No one has yet confirmed Fantham's observations, and there is no actual evidence that he was dealing with the schizonts of *L. lovati.* Moldovan (1914) described the asexual reproduction of *L. ziemanni* (Fig. 390, 12-14). The forms considered as schizogony stages were only seen in very heavy infections or acute attacks, which occurred just prior to death of the birds. Large cytoplasmic bodies up to 25 microns in diameter were found in the organs. The fully-developed schizonts contain a large number of nuclei, and segmentation into merozoites completed the schizogony process. Bodies of a similar nature were found by Coles (1914) in the blood and lung smears of an English thrush which harboured leucocytozoa. In this case the largest bodies measured 23·3 by 18·3 microns, and contained a large number of nuclei. Similarly, Knuth (1922) and Knuth and Magdeburg (1922) described the schizogony stages of a *Leucocytozoon* of geese in Germany, which they named *L. anseris*, as taking place in the internal organs either within mononuclear cells or free in the plasma. The description of these bodies as schizonts is in accord with what is known to occur in *Hæmoproteus*, and in view of the close relationship of the halteridia and leucocytozoa the forms noted by Moldovan, Coles, and Knuth and Magdeburg have probably been correctly interpreted as schizonts by these observers.

Nothing is known of the transmitting hosts of the leucocytozoa, but it will probably be found that these are the same as those responsible

for the transmission of the various species of *Hæmoproteus*, and it may be expected that a similar development will take place. From the researches of Danilewsky, Schaudinn (1904), Wasielewski (1908), the writer (1909), and Moldovan (1914), it is known that *in vitro* the gametocytes develop and give rise to oökinetes in the same manner as do gametocytes of members of the genera *Hæmoproteus* and *Plasmodium* (Fig. 382). The development up to this point will also take place in the stomach of *Culex pipiens*, as demonstrated by Schaudinn (1904), but it has not been proved that the mosquito is the transmitting host.

In this place can be considered a parasite which was described by Laveran (1902) as *Hæmamœba majoris* from *Parus major*. It differs from other leucocytozoa in the presence of pigment granules. Cardamatis (1911) claims to have seen pigmented forms of *L. ziemanni* in smears of bone marrow, and França (1912) noted pigmented leucocytozoa only once in fifteen infected birds (*P. major* and *P. cœruleus*). It seems, therefore, that pigment granules may occur exceptionally in certain leucocytozoa, but whether the pigment is of the same nature as that in *Hæmoproteus* has still to be determined.

As in the case of birds infected with halteridium, those harbouring leucocytozoa appear, as a rule, to be little affected by the parasite. Knuth and Magdeburg (1922) have, however, noted very heavy infections in young geese in Germany. The birds became seriously ill and died, and, according to these observers, this result was directly due to the leucocytozoa, which occurred in large numbers in all stages of development in smears of the blood and organs.

Wickware (1915) noted that sick ducks in Canada were heavily infected with a leucocytozoon, which he named *L. anatis*. Though healthy birds were not infected, and though the parasites disappeared from the blood of the sick ducks as they recovered, he hesitated to ascribe the illness to this particular organism.

2. *Family:* PLASMODIIDÆ Mesnil, 1903.

The members of this family, which are included in the single genus *Plasmodium*, are pigment-producing parasites which live in the red blood-corpuscles of vertebrates during the asexual cycle. Gametocytes are also produced in these cells. They develop no further in the vertebrate, and it is probable that in all cases they complete their sporogony cycle in invertebrates, which inoculate sporozoites into the vertebrates. The malarial parasites of man and birds are the only ones which have been completely studied, and in these cases the invertebrate hosts are mosquitoes.

The best-known members of the genus *Plasmodium* are the various malarial parasites of man and the closely allied malarial parasites (pro-

teosoma) of birds. On account of their supreme importance from the point of view of human disease, malaria of man being, perhaps, the most widespread and universal of all diseases, these parasites, since their discovery by Laveran (1880), have been the subject of most extensive investigations from every point of view. It may be claimed that their cycles of development are completely known, though certain details of this development and many of the factors which regulate it have yet to be elucidated.

Historical Account of the Discovery of the Malarial Parasite of Man, and its Development in the Mosquito.

Though pigmented leucocytes and pigmented bodies had been recognized in the blood and organs of persons who had had malaria by Meckel (1847), Virchow (1849, 1858), Planer (1854), and others, it was the most important discovery of Laveran (November 6, 1880) of the phenomenon of " flagellation " which convinced him that malaria was due to the invasion of the red blood-corpuscles by a pigment-producing protozoal parasite. Though Laveran's discovery was at first discredited by Italian and other writers, in spite of confirmation by Richard (1882), it was the Italians who eventually explained the asexual cycle and correlated the various forms which occur in the blood with the clinical picture of malaria as it is so well known to-day. Most important in this respect was the work of Golgi commenced in 1885, while much information was also contributed by other Italian observers, notably Marchiafava, Celli, Canalis, Grassi, Feletti, Bignami, Bastianelli. Sanfelice, and Manneberg. The problem of the ætiology of malaria still remained obscure, and though its possible transmission by mosquitoes had been suggested by Nott, Beauperthuy, King, and Laveran, the most precise prediction, and the one most accurate from the point of view of subsequent discovery, was that of Richard Pfeiffer (1892). This observer, after a study of the development of the coccidium of the rabbit, recognized the relationship of the malarial parasite and coccidia, and predicted for the former an exogenous cycle similar to that of coccidia, but which, owing to the absence of a resistant stage comparable with the oöcyst, might take place in the body of some blood-sucking insect. The resulting germ, he thought, might find its way into man through the sting of the insect, as Koch had suggested to him. It was Manson's observation that *Filaria bancrofti*, a disease-producing worm, the embryos of which occur in the blood-stream, developed in culex mosquitoes, which led him to the view, expressed in a paper published in 1894, that malaria would be found to be transmitted in a similar manner. So convinced was he of the ætiological connection of mosquitoes with malaria, that Ross determined to investigate the problem in India, with the remarkable and epoch-making results which have proved to be of such supreme importance from the point of view of preventive medicine. Manson's share in these investigations was duly acknowledged by Ross in the following words, with which he concludes his report (1898b): " These observations prove the mosquito theory of malaria as expounded by Dr. Patrick Manson; and, in conclusion, I should add that I have constantly received the benefit of his advice during the enquiry. His brilliant induction so accurately indicated the true line of research that it has been my part merely to follow its direction." Ross's experiments were no less remarkable than Manson's " brilliant induction," for he was working under difficulties and along entirely new lines. With precise information regarding the anatomy of mosquitoes, the methods of their

dissection, and what to expect after they have fed on malarial blood, experimental infection of mosquitoes and the study of the developmental cycle are comparatively simple matters at the present time. But when Ross was conducting his investigations every unexpected structure had to be explained, and the tracing of the life-cycle in the body of the mosquito was surrounded by obscurities, every one of which was overcome. Ross first discovered the life-cycle of the parasite of bird malaria in " grey mosquitoes," after having observed partially developed oöcysts of human malaria in " dappled-winged mosquitoes." Though he himself only saw immature oöcysts of the human malarial parasite in the mosquito, his prediction, which sub-sequent research has entirely justified, was that the human parasite would be found to have a cycle of development in the mosquito similar to that which he had described for the parasite of bird malaria. It remained for Italian workers to supply the proofs of this hypothesis and to raise it to the realm of demonstrated fact.

The actual part which Ross and Grassi played in these important discoveries has been the subject of endless controversy. In demonstrating the complete development of *Plasmodium præcox* in *Culex fatigans* and its transmission from bird to bird by this mosquito, Ross opened up an entirely new field, though it has to be remembered that Smith and Kilborne (1893) had already proved that a tick was responsible for the transmission of the parasite of red-water fever of cattle. Ross observed the early stages of the development of a human malarial parasite in mosquitoes, but failed to trace the complete cycle or to effect transmission of the disease. He, however, concluded that he had obtained sufficient evidence to justify his assertion that human malaria would be found to be transmitted by these insects—possibly by some particular species—in the same manner as bird malaria, and to advocate the destruction of mosquitoes as a method of prevention of the disease. Grassi may have been, and probably was, influenced and guided to some extent by what he had heard of Ross's discoveries, but nevertheless he and his co-workers were the first to obtain the absolutely scientific proof of the specific relation of anopheline mosquitoes to human malaria, and to follow the complete cycle of development of the three human malarial parasites in these mosquitoes, as Ross had done in the case of the parasite of birds.

Ross (1895) observed the flagellation of the crescent in the stomach of the mosquito, and in 1897 discovered pigmented cysts on the stomach of " dappled-winged mosquitoes " which had been hatched from pupæ and had fed on crescent-containing blood. In the following year (1898) he again reports the discovery of similar cysts on the stomach of a " dappled-winged mosquito." Ross (1898b) then turned his attention to bird malaria, and succeeded in elucidating the whole life-cycle of the parasite in culex mosquitoes. These results were first made public by Manson (1898), who announced Ross's discovery at the July meeting of the British Medical Association in Edinburgh, the proceedings of which were published on September 24, 1898.

An important advance had been made by MacCallum (1897), who, from observa-tions on the pigmented parasites (*Hæmoproteus*) of birds, discovered the true function of the "flagellating body," and identified the flagella as male elements destined to fertilize the female cell, which then became a motile vermicule. He also observed fertilization in the case of crescents of malignant tertian malaria.

After Ross's results with bird malaria had become known, it was not difficult for others to repeat and extend the work, as was done by various investigators in Italy, who were the first to prove that human malaria was transmitted by anopheline mosquitoes, and that the parasites had a cycle of development in these insects similar to that demonstrated by Ross for the para-site of bird malaria in culex mosquitoes. Bastianelli, Bignami, and Grassi (1898)

observed pigmented oöcysts in anopheline mosquitoes which had fed on a case harbouring crescents in the blood, as Ross had done in "dappled-winged mosquitoes" so early as August, 1897. These observers also succeeded in infecting a man with malaria by means of anopheline mosquitoes. Later in the year Grassi, Bignami, and Bastianelli (1898) observed the complete development of the malignant malarial parasite in *Anopheles claviger* (*A. maculipennis*), and the partial development of the tertian parasite. Early in the following year Grassi, Bignami, and Bastianelli (1899) reported the development of the parasite of quartan malaria in *A. claviger*, and Bastianelli and Bignami (1899) that of the tertian parasite in the same mosquito. They succeeded in infecting three men by bites of mosquitoes. Grassi, Bignami, and Bastianelli (1899a) then observed the development of the tertian and malignant parasites in *A. bifurcatus*, and Grassi (1899) that of the same parasites in *A. pseudopictus*. Ross (1899) found that the quartan parasite developed in anopheles in Sierra Leone. Bastianelli and Bignami (1899) published an illustrated paper describing the complete development of the tertian and malignant parasites in *A. claviger*, and showed that a single infected mosquito could transmit malaria to man. Ziemann (1900) in the Cameroons found that the malignant and tertian parasites developed in anopheles. Working in Holland, van der Scheer and van Berlekom obtained the development of the tertian parasite in *A. claviger*. Manson (1900) succeeded in infecting a man in London with malaria by means of anopheles imported from Italy, while Rees (1900) made a similarly successful experiment. Meanwhile, the work of Ross on the development of Proteosoma in *Culex fatigans* was confirmed by Koch (1899a) and Daniels (1899). The whole question was then placed on a sound basis by the publication by Grassi (1900) of a beautifully illustrated monograph describing in detail the complete life-cycles of the human malarial parasites in anopheline mosquitoes, and the importance of this particular type of mosquito in the epidemiology of the disease. This closed, as it were, a most interesting and important chapter in the history of scientific discovery in which the names of Laveran, Manson, Ross, and Grassi are specially prominent. Nuttall (1901) published a very useful account of the discoveries relating to mosquitoes and malaria, the papers dealing with the subject being chronologically arranged.

LIFE-CYCLE OF THE PARASITES OF MALARIA.

Cycle in the Vertebrate.—The vertebrate cycle of a parasite of malaria commences with the inoculation of sporozoites in the saliva which mosquitoes inject when they puncture the skin for the purpose of obtaining blood (Fig. 391, 32). The sporozoite thus introduced is a motile vermicule which, according to Schaudinn (1902a), forces its way into a red blood-corpuscle, where it soon becomes rounded off as a tiny mass of cytoplasm with a single nucleus (Fig. 392). Growth takes place at the expense of the cell, the parasite absorbing, not only nutriment, but also the hæmoglobin, which is transformed into a pigment (melanin, hæmozoin) deposited in the form of brown or black granules in the cytoplasm of the parasite. After a period of growth, which occupies two or three days, it is found that the single nucleus, by repeated divisions, has multiplied to form daughter nuclei, the number of which varies with the species. The parasite then produces a corresponding number of merozoites and a mass of residual

FIG. 391.—LIFE-CYCLE OF HUMAN MALARIAL PARASITE, *Plasmodium falciparum,*
IN MAN AND THE MOSQUITO (×ca. 1,000). (ORIGINAL.)

[*For description see opposite page.*

cytoplasm containing the pigment. By rupture of the red blood-corpuscle, which has been profoundly changed, the merozoites escape into the plasma. The residual body with the pigment is quickly phagocyted by leucocytes, but the merozoites attach themselves to other red blood-corpuscles, and by active movements similar to those shown by the sporozoites make their way into the cells, after which the process of growth and schizogony is repeated (Fig. 391, 1-7).

It has been maintained by Lawson (1913-1919) that the malarial parasites are not actually within the red cells, but on their surfaces, over which they move about, a view which was held by Laveran. It is also claimed that, during the growing period, the parasites may migrate from one cell to another. Sinton (1922c) justly points out that the mere inspection of the parasites in dried films in which the red cells have been flattened cannot decide the point as to whether the parasites are actually within or on the surface of the cells, though the marginal form is strongly suggestive of the latter view (Plate XII., 1-2, p. 926). He has attempted to settle the question by making the plasma hypotonic, so that the red cells swell, but do not burst. In the swollen condition he claims that it can be directly observed that the parasites, at least in their younger stages, are on the surface of the cells, and not within them. The evidence in favour of this view is not entirely convincing. If the parasites are actually extracellular, it is not easy to understand how the marked changes in the red cells, which occur during the growth of the parasites, are brought about, nor how it is that the red cell is completely destroyed when multiplication takes place. The appearance of the gametocytes in stained films and their behaviour when in the living condition in fresh blood certainly suggest an intracellular rather than an extra cellular habitat.

By repetitions of the schizogony process the number of organisms in the blood increases rapidly, till finally they are sufficiently numerous to

1-7. Schizogony cycle in red blood-corpuscles.　　　　8. Immature gametocyte.
　　9. Female gametocyte in blood of man.　　　　10. Male gametocyte in blood of man.
11-13. Development of male gametocyte and production of microgametes in stomach of mosquito.
14-16. Development of female gametocyte and extrusion of polar body in stomach of mosquito.　　　　17. Fertilization.
18-22. Transformation of zygote into oökinete and its penetration of the intestinal epithelium.
23-24. Development of oöcyst between the epithelium and the elastic membrane.
25-27. Growth of oöcyst coincident with multiplication of nuclei and vacuolation of cytoplasm.
　　28. Vacuoles have run together to reduce the cytoplasm to a coarse network, on the surface of which sporozoites are commencing to form as finger-like buds.
　　29. The sporozoites are fully formed and remain attached to several masses of cytoplasm into which the network has broken up. Two residual bodies containing nuclear material are present.
　　30. The detached sporozoites become irregularly distributed in the oöcyst, which ruptures, liberating the sporozoites into the cœlomic cavity.
　　31. The sporozoites enter the salivary glands.
　　32. Sporozoites injected into man by the mosquito, whence they invade the red blood-corpuscles and commence the schizogony cycle.

disturb the health of the individual. As the length of time required for the merozoite of any one species of malarial parasite to become a mature schizont is fairly uniform, it follows that all the organisms present at any one time are approximately at the same stage of development. The result is that when schizogony takes place, the vast majority of the organisms present are taking part in this process simultaneously, and it is then that the characteristic malarial attack occurs, as a result of poisonous substances liberated in the blood (Fig. 393). After the merozoites have again entered red blood-corpuscles the acute symptoms pass off, and do not recur till the next schizogony period is reached. On this account the symptoms reveal themselves at uniform intervals corresponding with the breaking up of the schizonts into merozoites. It is evident that if this development went on unchecked, a point would soon be reached where every available red cell of the blood would be used up. A condition of affairs approaching
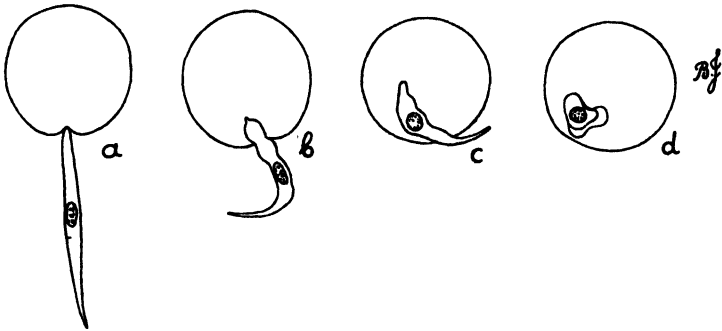
Fig. 392.—*Plasmodium vivax :* Penetration of Red Blood-Corpuscle by
Sporozoite (× 2,000). (After Schaudinn, 1902.)

this is seen in certain types of pernicious malaria, but, as a rule, the process is checked in some way, possibly through the production of antibodies in the blood, for the steady and uniform increase in parasites, which might be expected, does not take place.

To return to the life-cycle. After several generations of merozoites have been produced, certain of these, instead of growing into schizonts, develop into gametocytes (Fig. 391, 8-10). When mature, they are of about the same size as the fully-grown schizont, but contain more numerous granules of pigment, and have only a single nucleus. Two types can be recognized. The female-, or macro-gametocyte, has a dense and deeply-staining cytoplasm and a small compact nucleus. The male-, or micro-gametocyte, has a less dense and faintly staining cytoplasm, and a relatively large and diffuse nucleus. These gametocytes remain within the membrane of the red blood-corpuscles till they are taken up by the mosquito. If this does not occur, they eventually degenerate and die.

It has been claimed by Grassi (1900), Schaudinn (1902a), and some other observers, that the female gametocyte may, under certain circumstances, undergo a nuclear change, which converts it into a schizont capable of producing merozoites. This process, supposed to be one of parthenogenesis, was offered as an explanation of the relapses which frequently occur in malaria. It is well known that persons who have suffered from malaria, and who have been free from symptoms for long periods, may suddenly have attacks of malaria associated with the re-
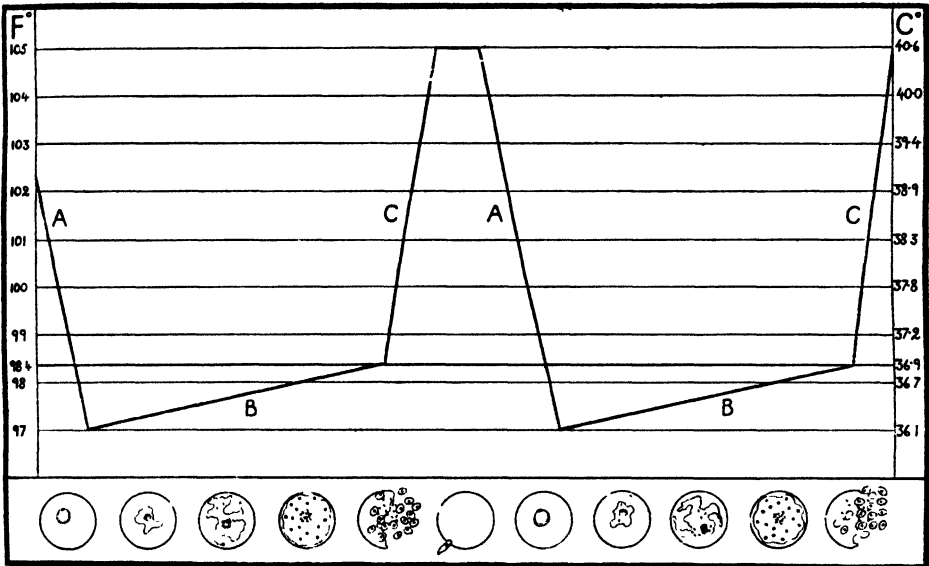
FIG. 393.—CHART OF TEMPERATURE IN MALARIA, SHOWING RELATION TO GROWTH AND SCHIZOGONY OF THE PARASITE. (ORIGINAL.)

The temperature (A) falls to subnormal when the merozoites have entered the red blood-corpuscles; B gradually rises to normal as the parasite grows; and C suddenly rises to produce fever when schizogony occurs and the merozoites and toxins are liberated into the plasma. The interval A to A, B to B, or C to C is forty-eight hours for *P. vivax*, seventy-two hours for *P. malariæ*, and forty-eight hours for *P. falciparum*; the interval C to A varies from six to twelve hours.

appearance of parasites in the blood, quite apart from the possibility of any reinfection by mosquitoes. Whatever may be the explanation of these relapses, they are unquestionably due to the presence of parasites, which, for some reason or another, have not been reproducing with their usual rapidity, probably owing to some controlling resistance on the part of the host. When this is relaxed, or, as it is usually expressed, when the vitality of the individual is lowered, the parasites commence reproducing in a more active manner. It does not appear that the supposed parthenogenesis of the female gametocyte explains the relapse any more clearly than the

more probable supposition that the usual asexual reproduction has been continued, but to such a slight extent that it produces no noticeable symptoms and escapes detection. Undoubtedly, many of the forms supposed to illustrate the parthenogenetic development of the female gametocyte can be explained as double infections of the cell (see p. 932). Mühlens, Weygandt and Kirschbaum (1920) inoculated the blood of a man which contained a large number of gametocytes of *P. falciparum*, but
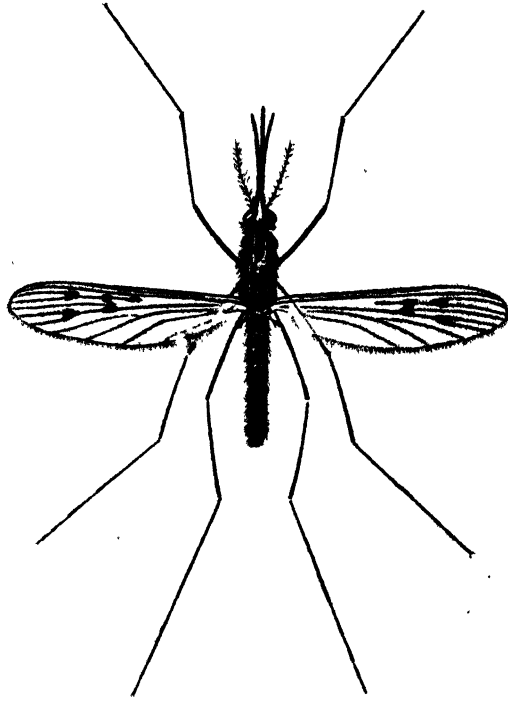
FIG. 394.—*Anopheles maculipennis* (♀), THE CHIEF TRANSMITTER OF HUMAN MALARIA IN EUROPE (× 6). (FROM BYAM AND ARCHIBALD'S *Practice of Medicine in the Tropics.*)

apparently no ring forms, into another individual. No infection resulted, though in other cases where ring forms were injected typical infections occurred. Lawson (1911) claims that the sexual cycle, which normally takes place only in the mosquito, may occasionally occur in the blood of man. The evidence put forward in support of this contention is far from convincing. It is logical, therefore, to accept the view that the gametocytes will ultimately degenerate unless they are taken up by the mosquito.

PLATE VII.

1-2. Formation of microgametes: " flagellating body."

3-4. Fertilization of macrogamete by microgamete.

5-7. Oökinetes in smear of stomach contents twelve and a half hours after feeding on case showing crescents in the blood.

8. Encysted zygote on stomach; pigment granules are adjacent to the nucleus, which has a large central karyosome.

9. First nuclear division in the zygote, the karyosome is dumb-bell-shaped.

10. Surface section of a later stage in development of oöcyst; the reticular character of the cytoplasm and one dividing nucleus are seen.

11-14. Sections through oöcysts during stage of growth and nuclear multiplication; the cytoplasm becomes more reticulated, while the nuclei, some of which show dividing karyosomes, become increasingly small.

[1-6. Dry films stained with Leishman stain; 7-13, sections of mosquito's stomach fixed in Schaudinn's fluid and stained with Mayer's acid hæmalum (7-11) or iron hæmatoxylin and eosin (12-13)].

(ORIGINAL.)

PLATE VII.

PLATE VIII

1

5

2

3

4

PLATE IX.

DEVELOPMENT OF *Plasmodium falciparum* IN ANOPHELINE MOSQUITOES : SPOROZOITES IN SECTIONS OF SALIVARY GLANDS.   (× 1,000).

1. Transverse section of lateral and central lobes of salivary gland.   In upper lobe the secretion is within the cells and the duct is small, while in the lower lobe the duct is much dilated by the secretion in which the sporozoites are embedded.

2 and 3. Longitudinal sections of lateral lobes of salivary gland, showing sporozoites in the cells and lumen of the duct.

(Fixed in Schaudinn's fluid and stained with Mayer's acid hæmalum.)

(ORIGINAL.)

PLATE IX.

1

2

3

Holmes f. 1910
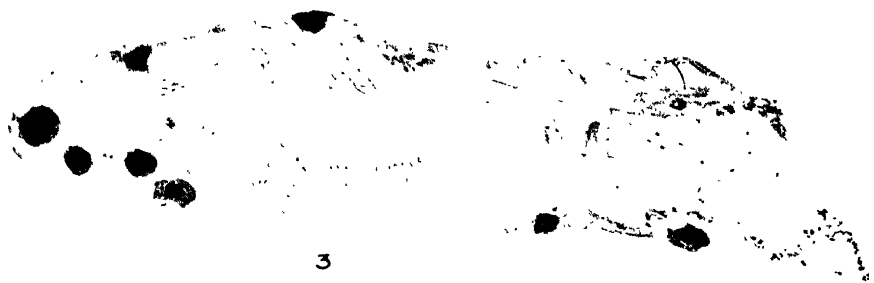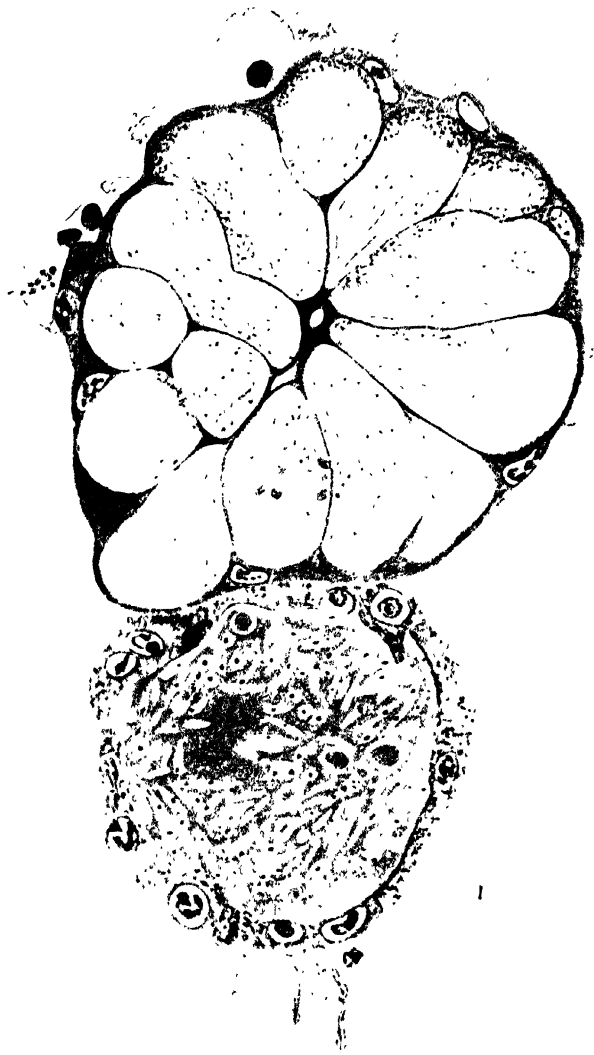
PLATE X.

Longitudinal section through salivary gland of *Anopheles maculipennis* showing sporozoites of *Plasmodium falciparum* in the cells and in the lumen of the salivary ducts (x *ca.* 1,000).

(Original compiled from six serial sections.)

PLATE XI
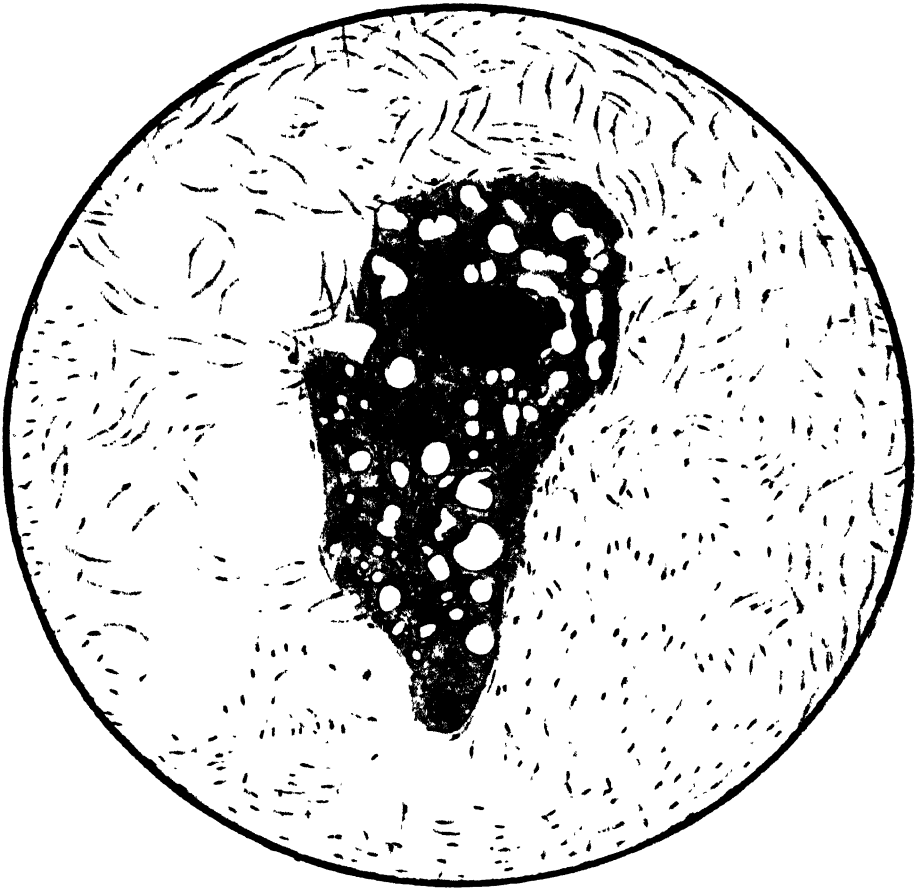
Smear of the salivary gland of a naturally infected *Phlebotomus* *perniciosus* caught in a house in Labunah, Macedonia (×1200). A single large gland cell is seen surrounded by numerous sporozoites. Dry film stained with Leishman stain

(Original)

Cycle in the Mosquito.—The gametocytes formed in the blood continue their development in mosquitoes. The earliest stages of development up to fertilization and the formation of oökinetes may occur in the stomach of any mosquito, as, indeed, in that of any blood-sucking insect, and on a microscope slide, but the further development of the oökinete will not take place in any but anopheline mosquitoes (Figs. 394, 395) in the case of human malarial parasites, and culicine mosquitoes in the case of those of bird malaria (Figs. 396, 397). When the mosquito feeds, it ingests not only gametocytes, but various asexual forms also. The latter degenerate and disappear with digestion of the blood, while the gametocytes alone develop further. By movements of contraction and expansion on the part of the gametocytes the membrane of the red blood-corpuscles is ruptured, and they escape. During this process the pigment granules in the male gametocyte are in a state of violent commotion. Its nucleus breaks up into a number of separate chromatin particles, and there are formed from its surface long thin processes of the cytoplasm, into each of which a chromatin particle enters (Fig. 391, 11-13). In this manner the microgameto-cyte may have attached to it from four to eight flagellum-like structures, which lash about continuously. This phenomenon gave rise to the term "flagellat-ing body." The flagellum-like structures are in reality the

FIG. 395. — *Anopheles maculipennis* (♀). SHOWING CHARACTERISTIC ATTITUDE WHEN RESTING ON A SURFACE (× 5). (ORIGINAL.)

microgametes. At varying intervals they break loose from the micro-gametocyte, the remnant of which constitutes a residual body, in which the pigment grains remain. It takes no further part in the life-cycle, and disintegrates. The liberated microgametes are actively motile, and by lashing movements swim about amongst the red blood-corpuscles in the mosquito's stomach. Meanwhile, the female gametocyte has been undergoing change. After escape from the red blood-cell it is spherical, but it does not show the active movements of the male gameto-cyte, while its pigment is stationary or only slightly motile. Frequently one or sometimes two minute bodies appear on its surface, and these are presumed to be derived from the nucleus, and to represent a maturation process comparable with the formation of polar bodies (Fig. 391, 14-16). The details of their formation have not been satisfactorily studied, and further investigations of the nuclear changes associated with fertilization are required. However this may be, the nucleus of the female gamete, as it now is, moves towards the surface, where a slight elevation of

the cytoplasm occurs.   Should a microgamete be in the vicinity, it enters
the summit of this elevation, and its nucleus ultimately unites with that
of the female gamete (Fig. 391, 17).   The whole of this process takes place
very rapidly, and may be studied in an ordinary wet blood-preparation
between a slide and cover-glass.   The formation of male gametes may
be completed within a few minutes of making the preparation, and
fertilization will then be taking place.   The zygote thus formed is for a
time a motionless sphere.   Soon it commences to elongate till, finally, a
vermicular form, which is motile, is assumed (Fig. 391, 18-20).
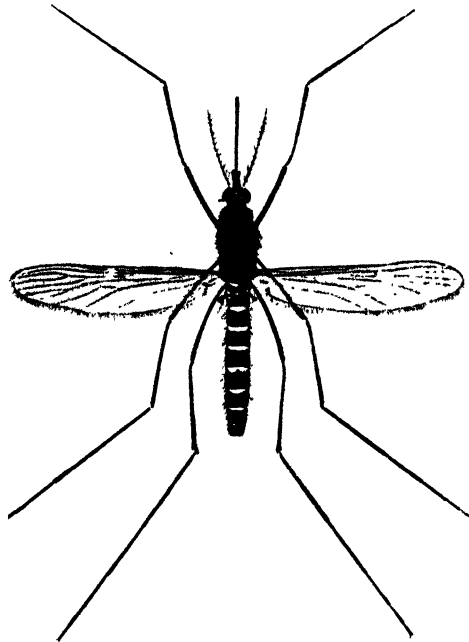
FIG. 396.—*Culex fatigans* (♀), THE COMMON TRANSMITTER OF BIRD MALARIA IN
INDIA (× 6).   (FROM BYAM AND ARCHIBALD'S *Practice of Medicine in the Tropics.*)

The exact process by which the microgametes arise at flagellation has
been variously described (Plate VII., 1-2, p. 916).   The nucleus of the male
breaks up into separate chromatin masses, the nuclear membrane disappear-
ing.   Some have supposed that the microgametes arise within vacuoles which
are formed in the cytoplasm, and that, by rupture of these, the microgametes
appear as flagella attached to the surface of the cytoplasm, where they lash
about till they break loose.   Another explanation is that finger-like processes
are rapidly formed at the surface, and that they become detached when

they have attained the required length. The most recent investigations are those of Brug (1916) on the formation of microgametes in *P. præcox* of bird malaria. He describes the formation of microgametes as taking place in the following manner. The cytoplasm at the margin of the male gametocyte, together with the nuclear material, spreads out into an exceedingly thin film. Spaces appear in the film, and reduce it to a series of narrow arches of cytoplasm containing nuclear material. The arches are attached to the body of the male gametocyte, or form anastomoses with one another. One end of each arch becomes detached, so that a series of filaments, attached to the main cytoplasmic mass at one end, is produced. If ordinary stained films of blood containing flagellating microgametocytes are made, appearances which seem to support this view may be encountered (Plate VII., 2, p. 916). The microgametes are often seen as a series of loops attached to the main mass of cytoplasm at both ends. Whatever may be the correct interpretation, the whole process takes place very rapidly, and is completed in a few minutes.

The significance of the flagellating bodies was long obscure, but MacCallum (1897), in the case of *Hæmoproteus* of birds and *Plasmodium falciparum*, followed the development up to the formation of the ookinete, and conclusively demonstrated the function of the flagella. He actually observed

the process of fertilization, and proved that they were microgametes. These observations have been repeated by other observers, including Schaudinn (1902*a*). According to this observer, the female gametocyte of *P. vivax*, ten to twenty minutes after entering the mosquito's stomach, undergoes maturation. The nucleus moves to the surface, and a small portion is separated as a bud with a little cytoplasm around it. Often two such bodies appear, but this is supposed to result from the breaking up of the single one at the time of its separation. After this, the nucleus retreats a short distance from the surface. The female gametocyte has now become the female gamete. Its fertilization takes place twenty minutes to two hours after the parasites have first been taken up by the mosquito (Plate VII., 3-4, p. 916). The nucleus of the male gamete, after its penetration, does not, however, fuse with that of the female for some time. The rounded body containing the two nuclei and the pigment granules, after a period of ten to twenty minutes' rest, puts out a clear cytoplasmic process or pseudopodium, which increases

in size till an elongated vermicule is produced (Plate VII., 5-7, p. 916). At the last moment of this process the vermicule or oökinete glides away from a gelatinous substance, to which are attached any male gametes which may have been adherent to the zygote, and also, not infrequently, some of the pigment granules. According to Schaudinn, a gelatinous substance is secreted on the surface immediately after entry of one microgamete, and serves to protect the female gamete from further interference. The oökinete, which measures from 18 to 24 microns in length by 3 to 5 microns in breadth, glides about amongst the intestinal contents. In the front region of its body is a vacuole behind which is the male nucleus, and further back still is the female nucleus. In the hinder region are the pigment granules. The male and female nuclei approach one another, and fusion occurs some hours after the first entry of the male gamete. The oökinete appears also to have increased somewhat in size. During its movements, the pigment granules, at first confined to the posterior regions of the body, may become distributed throughout the cytoplasm.

By movements of contraction, bending, and gliding the zygote or oökinete makes its way through the stomach contents towards the gut epithelium. It has an anterior and posterior end, a central nucleus, and the pigment granules of the female gametocyte in the post-nuclear region. Having reached the surface of the intestinal epithelium, it forces its way into a cell, through which it passes into the space between the epithelium and the elastic membrane which covers the body cavity or hæmocœle surface of the stomach (Fig. 391, 21-23). Between this membrane and the cells the oökinete comes to rest, and contracts to a small spherical body with a diameter less than that of a red blood-corpuscle. The pigment granules of the female gamete are still present in the cytoplasm. This migration mostly occurs during the second twenty-four hours after the feed of the mosquito. The earliest encysted zygotes may be slightly larger than the original macrogamete. The cytoplasm is homogeneous, and the nucleus is in a central position and consists of a nuclear membrane with a central karyosome (Plate VII., 8, p. 916). The zygote is soon found to be enclosed by a cyst, part of which, at least, appears to be derived from the elastic membrane (Fig. 391, 24). If, as appears probable, the elastic membrane is secreted by the cells in contact with which it lies, after it has been raised up by the parasite the cells may still continue secreting the membrane, so that a new portion will appear between the parasite and the cells. In this manner the zygote will become enclosed in a cyst composed of the elastic membrane. It seems probable that the cyst wall is formed partly in this way, and partly by the parasite itself secreting its own contribution to the cyst wall (Plate VII., 8, p. 916).

The zygote increases in size, the rate of growth depending on the temperature, till it has a diameter of 50 to 60 microns, the cyst wall stretching to accommodate it (Fig. 391, 24-27). That the cyst wall does not become appreciably thinner seems to indicate that the parasite is constantly adding to it. As growth of the zygote takes place, two changes occur. First, the nucleus multiplies by repeated divisions till an enormous number of minute nuclei is present. Secondly, the cytoplasm develops vacuoles, which gradually increase in number; they communicate with one another and with the space between the surface of the parasite and the cyst wall, till the whole of the cytoplasm is reduced to a sponge-work of numerous anastomosing septa (Fig. 391, 27-28, and Plate VII., 8-14, p. 916). This change brings about a great increase in the surface area of the cytoplasm, which is utilized by the nuclei in their subsequent development. The extent of vacuolation of the cytoplasm varies considerably. Sometimes very little takes place, and the sporozoites are formed only from the outer surface, and grow into the space between the cyst wall and the body of the parasite. Another modification sometimes seen is the presence of a single large central vacuole, from the surface of which sporozoites may or may not be formed. Usually, however, sporozoites are formed all over the cytoplasmic surfaces which have resulted from the vacuole formation. A section of an oöcyst at this stage will resemble a section of sponge. There will be areas of cytoplasm, which may or may not be connected with others.

When vacuolation is complete, the minute nuclei arrange themselves over the surfaces of the cytoplasm, and opposite each there soon forms a finger-like elevation which increases in length at the expense of the cytoplasm. Into each finger-like process a nucleus passes. In this way there are formed a number of narrow elongate sporozoites (Plate VIII., 1-2, p. 916). During the growth of the sporozoites the cytoplasm may break up into irregular masses to which the sporozoites are attached (Fig. 391, 28-29). It is important to note that these irregular masses are not sporoblasts, though they are often described under this name. When separate masses occur, they have been formed as described above, and are more the results of accident than of a definite process of development. They do not correspond in any way with the definite sporoblasts of coccidia, which are typically produced in uniform number within the oöcysts, and all of which subsequently develop in the same manner. The sporozoites, which are about 15 microns in length, eventually break away from their attachments and form a tangled mass within the oöcyst, which also contains one or more residual cytoplasmic bodies (Fig. 391, 30). In the latter the pigment grains may be detected, as well as one or more chromatin masses, apparently representing nuclei which have ceased to divide during nuclear multiplication.

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The number of oöcysts present on the stomach of any single mosquito varies with the number of gametocytes which were present in the blood on which it fed. Sometimes only one or two oöcysts occur. Very commonly ten to twenty are found, while not infrequently there may be thirty or forty, and exceptionally much larger numbers. In the case of *P. vivax*, the very heavy infections sometimes seen in *P. falciparum* do not occur, for in the case of the latter the gametocytes are often very much more numerous in the blood than they ever are in the case of *P. vivax* (Fig. 403). The cysts, which develop from parasites ingested at one feed,
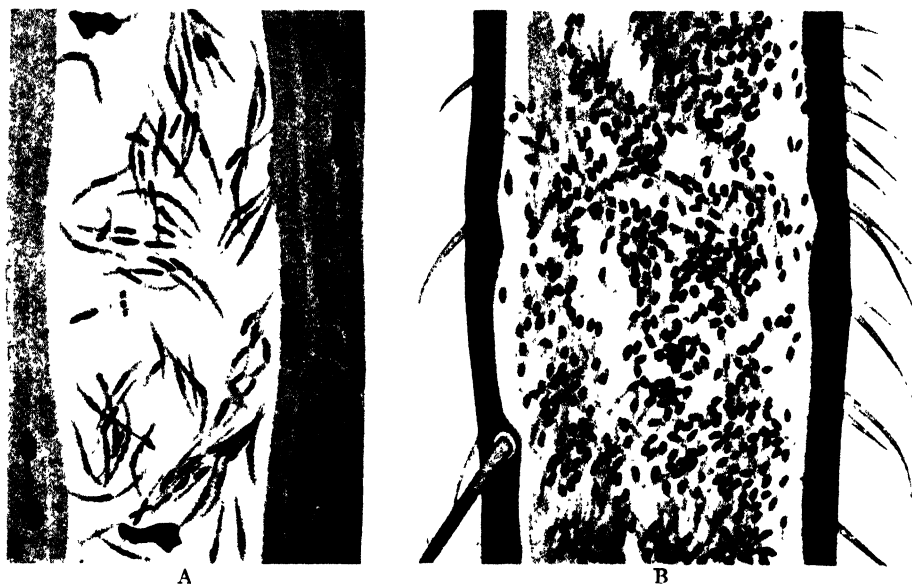
FIG. 398.—SPOROZOITES OF *Plasmodium vivax* IN BODY OF *Anopheles maculipennis* (× 1,500). (AFTER MÜHLENS, 1921.)

A. Between the fibres of a muscle.   B In a palp

are all at approximately the same stage of development at one time. If, however, the mosquito feeds repeatedly, then cysts of various stages of development will be present (Plate VIII., 5, p. 916).

The oöcyst, which is now mature, bursts and liberates the sporozoites into the body cavity or hæmocœle. They are pointed at each end, and have a central nucleus, which consists of a slightly elongated nuclear membrane and a central karyosome (Plate VIII., 4, p. 916). They are motile, and are able to progress by gliding movements, as well as by flexion and by waves of peristaltic constrictions which pass along the body. They wander through the body of the mosquito, and may be found in any organ. Mayer (1920), working with *P. præcox* of birds, and Mühlens (1921)

with *P. vivax* of man, have shown that the sporozoites may give rise to a veritable septicæmia in which masses of sporozoites occur in the aorta, between the muscle fibres, within the palps and scutellum, and in other situations (Fig. 398). Though the term septicæmia is employed, it does not mean that the sporozoites give rise to an infection which is fatal to the mosquito. Some of them, perhaps the majority, come in contact with the salivary glands which occur at the anterior part of the body cavity (Fig. 391, 31). They penetrate the cells of the glands, and may be found in enormous numbers embedded in the cytoplasm (Plates IX. to XI., p. 916). As the saliva is secreted into the salivary duct, sporozoites pass with it, and are finally injected into the skin by the mosquito at the moment of biting. They enter the red blood-corpuscles and recommence

1   2   3

FIG. 399.—OÖCYSTS OF MALARIAL PARASITE AND THE "BLACK SPORES" IN *Anopheles maculipennis* AFTER FEEDING ON MALARIAL CASES (×1,000). (1 AND 2, FROM PREPARATIONS MADE BY COLONEL JAMES; 3, FROM MOSQUITO INFECTED BY THE WRITER IN MACEDONIA.) (ORIGINAL.)

    1. Intact cyst containing spores (*P. vivax* infection).
    2. Isolated spores of smaller size from another mosquito (*P. vivax* infection).
    3. Isolated spores from another mosquito (*P. falciparum* infection).

the asexual cycle (Fig. 391, 32). As the sporozoites themselves cannot reproduce any further in the mosquito, it follows that a time will arrive when all the sporozoites will have been removed.

Black Spores. In his experimental work on the development of *P. præcox* in *Culex fatigans* in India, Ross noted that occasionally peculiar brown or black cysts appeared on the stomach of the mosquitoes in the place of the normal ones. The colour of these was due to certain dark bodies within the cysts. The same cysts were afterwards seen by Grassi during his work on the development of the human malarial parasites in anopheles (Fig. 399). He noted that the bodies within the cysts were either sausage-shaped, rounded, oval, or irregularly lobed. Each was composed of minute brown granules embedded in a clear substance which was not enclosed by any membrane. Various suggestions have been made as to the nature of these black spores. That they are spores of a

microsporidian cannot be entertained, as they do not bear any resemblance to these. They are probably the result of death and degeneration of the oöcyst contents at various stages of its development. It is possible that chitinous material is deposited in them by the mosquito.

The life-cycle just described applies to the parasite of bird malaria, which develops in species of *Culex*, and to those of human malaria, which develop in species of *Anopheles*. As noted above, *Hæmoproteus columbæ* of the pigeon has a similar developmental cycle in the hippoboscid fly, *Lynchia maura*. A number of other species of *Plasmodium* are known. They have been studied only in the blood of the vertebrate, and in no case has the transmitting host been discovered.

SYSTEMATIC DESCRIPTION OF SPECIES OF PLASMODIUM.

The family Plasmodiidæ contains the single genus *Plasmodium* Marchiafava and Celli, 1885. Schizogony takes place within the red blood-corpuscles of the vertebrate host, while during growth of the parasite a pigment (hæmozoin) is formed from the hæmoglobin of the cell. The gametocytes, which also occur in the red blood-corpuscles and contain pigment, undergo their further development (sporogony) in mosquitoes. Microgametes are produced by an active process of flagellation, while the zygote becomes a motile oökinete before encystment in the oöcyst. The latter increases enormously in size before sporozoites are formed. There is no formation of sporoblasts or sporocysts.

PLASMODIA OF MAN.

Human beings are liable to infection with three well-established species of malarial parasite (Plates XII and XIII., pp. 926, 934). They all belong to the genus *Plasmodium*, in spite of the fact that some observers have maintained that the parasite of malignant tertian malaria, the gametocytes of which are crescent-shaped, should be placed in a separate genus, *Laverania*. The life-cycles of the three forms (*P. vivax*, *P. malariæ*, and *P. falciparum*) resemble one another very closely. Other species have been described, but there is no evidence to indicate that the forms on which they were based are not merely abnormal individuals of the well-known types.

The disease produced by these parasites is characterized by attacks of fever, which commence at the moment the merozoites resulting from schizogony escape from the red blood-corpuscles into the plasma. When the merozoites have again entered other corpuscles, the fever subsides, only to recur when schizogony is repeated (Fig. 393). It is probable that the

fever is actually caused by toxic substances which escape from the ruptured corpuscles, and that the gradual enlargement of the spleen, which is a constant feature of chronic malaria, is due to the continued irritation by the same toxins. The repeated destruction of red blood-corpuscles induces a profound anæmia, which is associated with an increase of the large mononuclear cells in the blood, while special symptoms referable to the accumulation of parasites in the vessels of particular organs may supervene.

Plasmodium vivax (Grassi and Feletti, 1890).—This organism causes the disease of man known as benign tertian malaria (Plate XII., p. 926). It is called benign because it is rarely fatal, and tertian because the attacks occur every third day, the parasite requiring forty-eight hours to complete its asexual cycle and reproduce by schizogony. The parasite was first recognized as a distinct species and separated from the one producing quartan fever as a result of the researches of Golgi (1886).

The Cycle in Man.—The infection is commenced by the entry of a sporozoite into a red blood-corpuscle, a process which has been seldom observed. Schaudinn studied the behaviour of sporozoites by injecting them into a small cutaneous hæmatoma and abstracting blood from it by means of a pipette. He noted that the sporozoite approached the red blood-corpuscle end on, indented it at the point of contact, and by active movements gradually forced its way into the stroma (Fig. 392). At first slightly elongate, it soon retracted to form a disc of cytoplasm with a more or less circular outline. The merozoites subsequently formed at schizogony are comparatively short and thick, while the sporozoites are long and slender. The merozoites enter the red cells in much the same way as do the sporozoites. As Schaulinn points out, the mode of entry of the sporozoites and merozoites into the red blood-corpuscles gives no support to the view, noted above, that the malarial parasites are actually parasitic on the surface of the cells. In either case, the resulting organism in the cell has the form of a disc of cytoplasm with a single nucleus. Very soon a vacuole appears, causing the tiny nucleus of the parasite to be pushed to one side. It then has the appearance of a ring, and is known as the signet ring form. It has a diameter of about one-third of that of the red blood-corpuscle (Plate XII., 3-5, p. 926). In blood-films of heavy infections, in addition to the ring forms young forms may be seen at the edge of the red cells (marginal forms). They appear either as little streaks of blue cytoplasm with a red nucleus (flattened marginal forms), or if the vacuole has developed, the red cell is slightly indented at this point, and the cytoplasm not only lines the margin of the concave indentation, but forms an arch from one end of the indentation to the other, with the chromatin dot at some point of the arch (Plate XII., 1-2, p. 926). These forms are actually disc-shaped and have a large

central vacuole, but on account of their arched appearance are called bridge forms. It must be remembered that the ring form is that of the resting condition, for if the organism be observed on the warm stage, it will be seen constantly to change its shape, throw out pseudopodia, and exhibit amœboid movements. The amœboid activity is very characteristic of all the stages of growth of the schizont, and gives rise to the name *P. vivax*. In the ordinary method of making dry blood-films, all the pseudopodia, especially of the youngest forms, are withdrawn, so that the youngest parasites are most usually seen in stained films as the typical ring forms. Occasionally, however, very irregularly shaped amœboid forms of the youngest stages are seen in stained films. By absorption of nutriment, which is rendered the more easy because of the increased surface resulting from the formation of the large vacuole, the parasite increases in size, and after about five or six hours' growth it either has a more irregular shape (amœboid form), or the ring form (large ring form) is still maintained, and one or two refractile granules of yellow or light brown pigment will have been deposited in the cytoplasm (Plate XII., 6-10). The vacuole is also larger, but has not increased to the same extent as the cytoplasm. At this stage, three changes will be seen to have taken place in the red blood-corpuscle. It has increased in diameter, has become slightly paler, and has developed on its surface a number of fine granules, which stain red by Romanowsky stain. These granules are known as Schüffner's dots. They may not be visible in poorly stained films, and it is important to remember this point in forming opinions as to their presence or absence. These three changes in the infected red blood-corpuscles, which become more marked with continued growth of the parasite, are characteristic of *P. vivax*, as compared with the other malarial parasites of man, and are of great importance from a diagnostic standpoint. As growth continues, the parasite becomes more irregular in shape owing to the formation of long thin pseudopodia, which extend through the stroma of the cell in various directions and form anastomoses with one another. The parasites maintain their irregular shape in ordinary dried films, probably because they are too large to completely withdraw their pseudopodia, as the smaller forms usually do. In films that have dried slowly the shape is more compact. The parasite continues to increase in size for about thirty-six hours, after which period it has the form of a coarse irregular meshwork, which may assume any conceivable shape. Numerous yellowish-brown pigment grains are scattered through the cytoplasm. The nucleus, which in the younger stages was composed of a fine membrane enclosing a relatively large karyosome in the form of a compact mass of chromatin staining material, has now increased in size, and possesses several granules distributed through the space enclosed

PLATE XII.

Plasmodium vivax, THE PARASITE OF BENIGN TERTIAN MALARIA OF MAN, AS SEEN IN DRIED BLOOD-FILMS STAINED WITH ROMANOWSKY STAIN. (× 2,000).

1. Marginal young form.
2. Arched young form.
3-5. Young ring forms.
6-11. Growth of schizont—enlargement of red cell, formation of Schüffner's dots, and development of pigment in cytoplasm of parasite.
12-18. Nuclear multiplication and schizogony.
19. Double infection with schizont and gametocyte.
20. Double infection with two gametocytes.
21-25. Growth of gametocyte.
26. Female gametocyte (macrogametocyte).
27. Male gametocyte (microgametocyte).
28-29. Multiple infection with young ring forms with fused cytoplasm (" tenue phase " of Chalmers and Archibald).
30. Cell containing a nearly mature gametocyte and young form.
31-35. Unusual type—absence of Schüffner's dots and little or no enlargement of red cell.

(ORIGINAL.)

PLATE XII.

B. Jobling
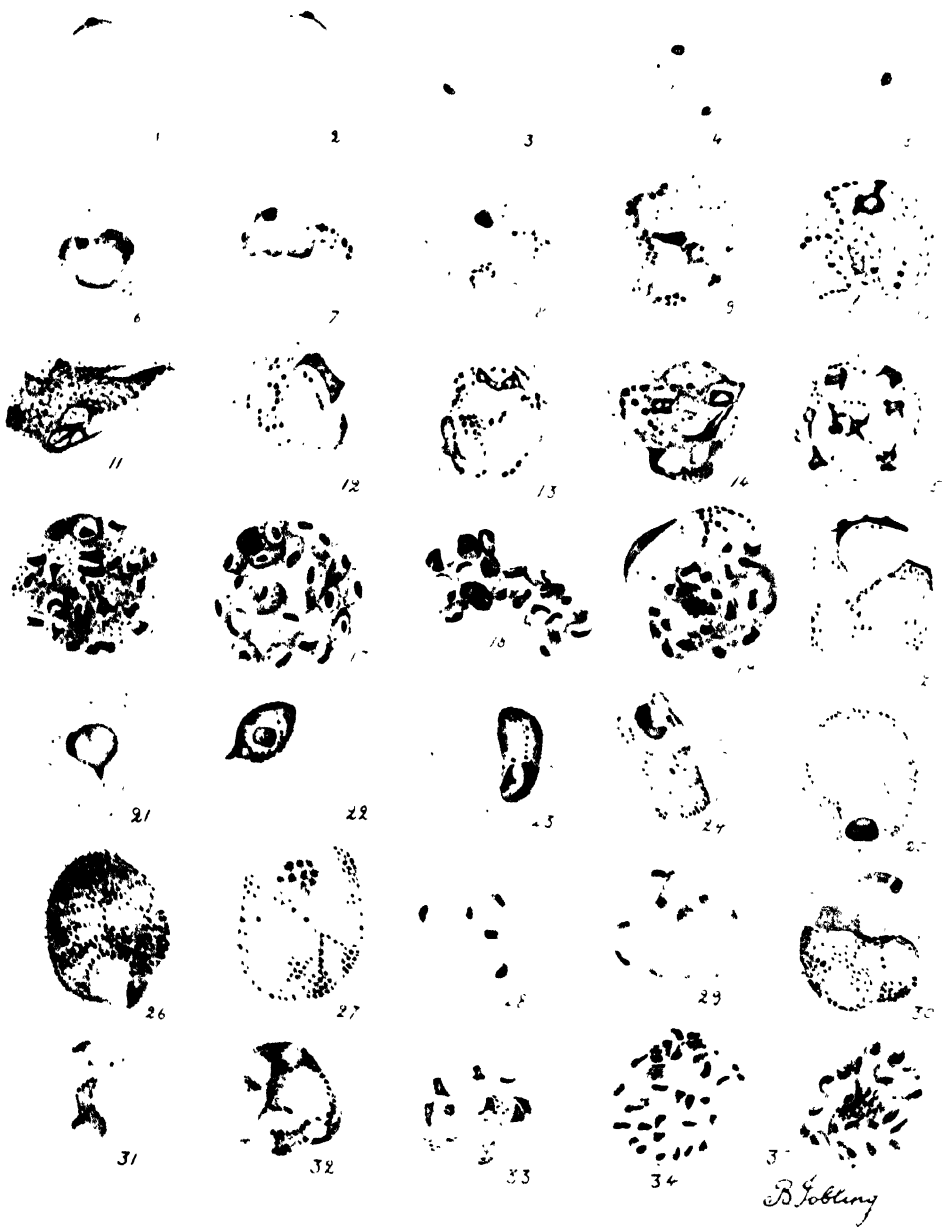
by the membrane. The cytoplasm of the parasites extends through the now distinctly enlarged red cell, which in properly stained films is seen to be dotted over with numerous Schuffner's dots. After thirty-six hours growth proceeds more slowly, and nuclear changes preparatory to nuclear division, which was studied in detail by Schaudinn (1902a), occur. The chromatin granules become smaller, and the parasite itself assumes a more compact form, with merely an irregularity in its outline. Nuclear division, as seen in properly fixed films, is initiated by the fine chromatin granules of the nucleus collecting as a band or equatorial plate across the diameter of the nucleus (Fig. 400). This plate then divides into two plates, which separate from one another. Division of the nuclear membrane follows, and the granules of each plate form the chromatin of the daughter nuclei. Each daughter nucleus then divides in a similar

FIG. 400. Plasmodium vivax. FIRST NUCLEAR DIVISION IN SCHIZONT BY MODIFIED MITOSIS (× 2,000). (AFTER SCHAUDINN, 1902.)

manner, as do the four resulting nuclei. After this stage, nuclear division proceeds more irregularly, and Schaudinn was not able to observe the equatorial plate formation any further. In the subsequent divisions, the chromatin granules of each nucleus are divided into two more or less equal groups. Nuclear divisions take place till typically sixteen nuclei are present. The number, however, is by no means constant, as the number of nuclei varies from twelve to twenty-four, the irregularity in number being due to the fact that some of the daughter nuclei cease to divide (Plate XII., 11-15). While nuclear multiplication has been taking place the parasite has become larger, till after forty-six hours' growth it has the form of a circular plate of cytoplasm, which almost completely fills the enlarged red cell. The latter has increased in diameter from 7 to about 10 or 11 microns, and in stained films is still seen to possess

Schüffner's dots, which are relatively larger and more numerous than in the more recently infected cells. The cell is very pale in colour when compared with uninfected cells, while the parasite contains about fifty pigment granules, which, as nuclear multiplication reaches completion, collect together in one or more aggregations. The chromatin granules of each nucleus now form a more compact body, which appears to lie in a small vacuole, the limits of which undoubtedly represent a very fine nuclear membrane. After this the cytoplasm, by a process which is actually one of budding, gradually segments into a number of portions around the nuclei, and the merozoites are separated, leaving the pigment in one or more masses of residual cytoplasm (Plate XII., 16-17, p. 926). The merozoites remain for some time in the red cell, which has been reduced to little more than a membrane. The latter bursts, and the merozoites are scattered into the plasma, when they quickly attack other cells, enter them, and repeat the process of growth as just described (Plate XII., 18, p. 926).

The pigment which is liberated when the corpuscle ruptures is taken up by various leucocytes, but especially by the large endothelial cells of the capillaries, the majority of which are *in situ*, but some of which have become detached and are circulating in the blood as large mononuclear leucocytes. It is a feature of malarial attacks that the number of these cells free in the blood is increased. Pigment granules may sometimes be found in these circulating cells (pigmented leucocytes) when actual parasites cannot be found in the red cells.

Though the majority of the parasites attain maturity at about the same time, this is not true of all. Some attain maturity before and others after this period, the resulting merozoites again attaining maturity earlier or later than the majority. It comes about, therefore, that schizogony may actually occur at any period. The minimum amount of schizogony will be taking place when the majority of schizonts are half grown (twenty-four hours), and the nearer the critical period is approached (forty-eight hours), the greater will be the amount of schizogony actually occurring. In any case, the vast majority of parasites attain maturity at or near the critical period of forty-eight hours (Fig. 393). The irregularity just noted must be distinguished from that resulting from what are called double infections, which are attributed, possibly without sufficient evidence, to an individual having been inoculated with sporozoites on two successive occasions at a twenty-four hour interval. In such cases there will be two distinct batches of parasite present, one always twenty-four hours behind the other as regards age. There will then be two critical periods at twenty-four hour intervals, the schizonts of one batch attaining maturity on one day and those of the other twenty-four hours later. The

result is that malarial attacks occur daily instead of on alternate days. It is generally recognized that attacks occur most usually in the forenoon, and, as presumably the sporozoites are inoculated at night, it is clear that the periods of growth must have been either more or less than forty-eight hours in order to bring this about. There is evidently some factor which favours schizogony at this particular time of day, and it is easy to imagine that the parasites might become grouped in two batches, one of which, by hastened development, would reproduce twenty-four hours before the other. The factor or influence affecting the parasite, if it depends on the host, would be expected to repeat itself every twenty-four hours. If this be correct, then the appearances of a double infection might be produced from a single batch of inoculated sporozoites. Recent work in connection with the treatment of general paralysis by inoculating malaria is tending to prove that double infections can be produced in this manner.

After a mosquito has injected sporozoites, the initial attack of malaria occurs in about ten to twelve days. That is to say, it requires about five successive cycles to produce a sufficient number of parasites to give rise to symptoms. Assuming that at each cycle fifteen merozoites are produced, and that all these develop to maturity, by a simple calculation a single sporozoite will be seen to give rise, in this case, to over 759,000 parasites.

After schizogony has been repeated a number of times gametocytes appear in the blood. Usually they do not occur till about ten to fourteen days after the initial attack, but they have actually been noted at the time of the first attack which occurred. Bastianelli and Bignami (1899) were able to infect mosquitoes from a case of malaria on the fifth day of the illness, which resulted from an experiment with infected mosquitoes. The gametocytes are developed from merozoites, which grow in a different manner to those producing schizonts. According to Schaudinn (1902a), after entering a red blood-corpuscle, they become rounded off as compact bodies which do not develop a vacuole, and so do not assume the signet ring form. Though the earliest stages of this development can be seen in the peripheral blood, for some reason not properly understood the growth of the merozoite into the gametocyte takes place almost entirely in the vessels of the spleen or bone marrow, owing to the red blood-corpuscles containing them ceasing to circulate, and becoming held up in these organs. Only exceptionally are the immature gametocytes seen in the blood-stream (Plate XII., 21-25, p. 926). In consequence of the small surface area due to the lack of the vacuole, growth proceeds much more slowly than in the case of the schizont. Furthermore, the active amœboid movements are not seen, the parasite growing steadily as a

rounded compact body. On the warm stage, however, slight alterations in shape can be seen to take place. The effect on the red blood-corpuscle is the same as that produced by growth of the schizont. There is a marked increase in size, development of Schüffner's dots, and loss of colour. The gametocytes attain maturity in about ninety-six hours. They are of two kinds, the male and female gametocytes, which may be distinguished from one another. The female (Plate XII., 26, p. 926) has a denser cytoplasm, which stains deeply blue with Romanowsky stains, owing to accumulation of food material, while the male (Plate XII., 27, p. 926) has a more hyaline cytoplasm, which stains a very pale blue. The nucleus of the female is small, and consists of a nuclear membrane, within which is either a single karyosome or a group of granules. The male nucleus, on the other hand, is large, its chromatin granules are fine, and irregularly distributed within the nuclear membrane. Both the male and female gametocytes contain the yellowish-brown pigment granules distributed through the cytoplasm, and there is a greater number present than in the mature schizonts. As remarked above, when the schizont nears maturity, the irregular form characteristic of its earlier stages is not so manifest. The organism becomes more nearly circular in outline, and difficulty is sometimes experienced in distinguishing the gametocytes, which are about the same size and also circular in outline. The gametocytes, however, are more regularly circular in outline; they contain a larger number of pigment granules, which are more or less uniformly distributed through the cytoplasm, and the nucleus is always single, whereas in the schizonts of equal size the outline is not so regularly circular, the smaller number of pigment granules are already becoming aggregated into one or more groups, while the nucleus has already multiplied, preparatory to schizogony, so that several, at least, are present. In cases of doubt, an observation of a fresh blood-preparation under the microscope will reveal the presence of gametocytes, for these will develop further, the male gametocytes producing microgametes by the characteristic process of flagellation, and the females becoming contracted and separated from the host cell in readiness for fertilization.

Types of Parasite in Blood-Films made at Intervals.—In the case of single infections where only one generation of parasite is present, the following appearance will be noted in blood-films made at different periods of the cycle (Fig. 393): At the time of an attack of malaria a blood-film will show schizonts nearly mature, schizonts which are mature and in actual process of breaking up into merozoites; ruptured red cells discharging merozoites, and consequently free merozoites in the plasma, either in groups or separately; merozoites adherent to the margin of red cells and merozoites within the red cells, some of which have no vacuole,

and others, a little older, which have developed the vacuole, and consequently show the typical signet ring form. In addition, mature male and female gametocytes are present. Very young gametocytes may also occur as solid forms without a vacuole, but these are hardly distinguishable from the youngest schizonts, which have not yet developed a vacuole. Shortly after an attack all the schizonts will have broken up, so that the only forms present will be ring forms and mature gametocytes, and possibly some young gametocytes. A very occasional schizont may, however, be found. About twenty-four hours after an attack there will be present numbers of half-grown irregular schizonts, and again mature and possibly some young gametocytes. A very occasional mature schizont may, however, be encountered. The rare occurrence of mature schizonts between the attacks has been explained above. It will result, therefore, that these rare schizonts will be producing merozoites whenever they attain maturity, so that ring forms and partially grown or mature schizonts may be found at any time in very small numbers. Twelve hours later or thirty-six hours after an attack the schizonts will be still larger, and some of them will show indications of nuclear multiplication in the presence of two or four chromatin masses. Gametocytes in various stages will still be present, as also occasional ring forms or mature schizonts. Shortly before the next attack, or about forty to forty-five hours after the previous one, fully-grown schizonts with numerous nuclei and concentrated pigment will be found. Gametocytes are present as before, while occasional asexual forms at any stage of development may also be present.

In the case of double infections at the time of an attack there will be present all the forms seen in the single infection, together with a large number of half-grown schizonts and mature gametocytes. Twenty-four hours later all the half-grown schizonts will have become mature; consequently, there will be another attack, and the same forms will be present as at the first attack. The mature schizonts and ring forms are, however, those which were only half-grown at the first attack, while the half-grown forms are those which were ring forms or mature schizonts on that occasion. As in the case of single infections, irregularly appearing ring forms, partially grown forms, and schizonts may occur in very small numbers at any stage of the infection.

Intensity of Infection.—The number of organisms present in the circulating blood at any time varies considerably, and there is evidence that even when a large number is present, still larger numbers occur in the vessels of the spleen and bone marrow. Just after an attack of fever, when schizogony is complete, practically all the asexual forms are in the ring stage. In heavy infections, a large proportion of the red blood-corpuscles will be

parasitized, while some cells contain two or even three or four rings. The number of rings present, however, never reaches the enormous number sometimes seen in heavy infections with *P. falciparum*, in which multiple infections of individual cells is quite common, whereas it is the exception in the case of *P. vivax*. There is no relationship between the number of gametocytes which may be present and the other forms. Sometimes large numbers of the asexual forms are present, and very few gametocytes can be found. At others the gametocytes are comparatively numerous, while the asexual forms are few in number or cannot be detected at all. Furthermore, the gametocytes present may at one time show a preponderance of male forms, and at another a preponderance of female forms. The general rule, however, is for fairly numerous asexual forms to be present, while a smaller number of gametocytes, both male and female in approximately equal numbers, occurs at the same time.

In the case of individuals who have not had malarial attacks for long periods, prolonged examinations of the blood will sometimes reveal isolated parasites at any stage of development. The occurrence of these seems to be an indication that the cycle of development has been continued throughout this period to such a slight extent, owing to some controlling influence, that the parasites never reach the number sufficient to produce the typical malarial attack. It is probable that the developmental cycle of the parasites in these latent infections takes place almost exclusively in the vessels of the spleen and bone marrow, and that it is only rarely that isolated forms are found in the peripheral blood. Every now and then the balance which had been struck between the host and the parasite is lost, and active multiplication is resumed, with the result that a typical attack supervenes. This seems to be the most reasonable explanation of relapses occurring at long intervals, but, as explained below, another explanation has been given. The gametocytes, when mature, do not develop further till they reach the stomach of the anopheline mosquitoes. If this does not take place, they degenerate and disappear, the more delicate males first, and the females later.

Supposed Reproduction by Parthenogenesis.—Grassi (1900) and Schaudinn (1902a) observed certain changes in the female gametocytes of *Plasmodium vivax*, which were interpreted as a process of parthenogenesis. Schaudinn noted them in the blood of cases shortly before a relapse after an interval of freedom from attacks. The nucleus of the female gametocyte becomes elongated and divided into two parts, one of which contains coarse chromatin granules and the other fine ones. The coarsely granular nucleus then proceeds to divide by repeated divisions exactly like the nuclear multiplication of the schizont. When about sixteen nuclei are present, a segmentation into a number of daughter

individuals or merozoites takes place. A certain amount of residual cyto-plasm is left over, and this contains the pigment and also the nucleus with the fine chromatin granules. The merozoites thus formed infect other red cells, and proceed to develop into schizonts. In this process it is supposed that the division of the nucleus into two, which first takes place, is a means of ridding the female gametocyte nucleus of its preponderating sexual quality, the nucleus which is cast off with the residual body con-taining the sexual chromatin, while that which proceeds to the formation of the merozoite nuclei contains the asexual or vegetative chromatin. The writer has long held that the appearances from which this partheno-genetic process was deduced were wrongly interpreted by Schaudinn and others who have followed him. It frequently happens that a red blood-corpuscle is infected with two or more parasites at the same time. The two may be both young schizonts, and as they attain maturity the outline between the two is difficult to distinguish, so that when schizogony actually occurs there is produced the appearance of a very large parasite producing thirty or more merozoites. In other cases, a cell may be infected with a merozoite which is to become a schizont and with another which is to become a gametocyte. Here, again, the line of separation, especially in dried films, may be difficult to detect, so that when the schizont proceeds to break up into merozoites the two parasites appear as one large parasite breaking up into merozoites on one side only, while at the other side there is a mass of cytoplasm, actually the gametocyte, containing an undivided nucleus (Plate XII., 19, p. 926). On other occasions, a cell may be found containing two male or two female gametocytes or a male and female gametocyte (Plate XII., 20, p. 926). Practically all conceivable com-binations may occur, and it seems to the writer that the figures given by Schaudinn to illustrate his process of parthenogenesis are more reasonably explained on the basis of double infections of cells by a schizont and gametocyte. Thomson (J. D.) (1917) came to an exactly similar con-clusion, and refuted the whole of Schaudinn's theory of parthenogenesis as applied to malarial parasites. Attempts have been made by Pontano (1920) and Samsonoff (1925) to revive the doctrine, but it must be admitted that the figures given by them are far from convincing, and even if it be admitted that the nature of the parasites depicted cannot be completely explained, there is still no particle of evidence that the process is one of parthenogenesis any more than one of degeneration.

The Cycle in the Mosquito.—As regards the development of *P. vivax* in mosquitoes, there is little to add to the general description given above (p. 914). It is possible that, as the gametocytes are the largest, the zygotes and oöcysts of *P. vivax* are also larger than those of the other species. This is the writer's impression, but he has no precise measure-

ments to support it. In one respect, however, the various species can be distinguished. The pigment produced by *P. vivax* is of a light brown colour, while that formed by *P. malariæ* and *P. falciparum* is dark brown or even black. It follows, therefore, that zygotes and oöcysts with light brown pigment belong to *P. vivax*. Furthermore, the pigment granules are fine, and are not infrequently arranged in a curved line instead of in an irregular clump. The rate of development of *P. vivax* in mosquitoes differs from that of *P. falciparum*, but this aspect of the question is discussed below (p. 958).

Plasmodium falciparum (Welch, 1897).—This is the organism which causes malignant tertian malaria of man (Plate XIII., 16-40, p. 934). Golgi (1886) was the first to suspect that it was a distinct species, and suggested that the crescents belonged to a cycle of development which differed from those of the parasites of benign tertian and quartan malaria. In a later paper (1889) he definitely asserted that, in addition to the tertian and quartan fevers, a third type associated with crescents had to be recognized. Later in the year Marchiafava and Celli (1889) gave the first clear account of the differential characters of the malignant tertian parasite. This was followed by a similar description by Canalis (1889), and a fuller one by Celli and Marchiafava (1890). As with other malarial parasites, there is great confusion as to the correct name of this one. Much depends on whether the organism should be in a genus distinct from the other malarial parasites on account of the crescent shape of its gameto-cytes. Grassi and Feletti (1890) proposed the new generic name *Laverania*, and named the parasite *L. malariæ*. As Schaudinn (1902a) pointed out, the mere difference in shape of a gametocyte is not in itself sufficient to justify a separate generic name. The correspondence in the life-cycle is so close that it seems clear that the parasite of malignant tertian malaria must be included in the same genus as the other malarial parasites of man. It cannot have the name *P. malariæ*, for this has priority for the parasite of quartan malaria. Grassi and Feletti (1892) had employed the name *Hæmamœba immaculata* for a form of malarial parasite which they considered to be devoid of pigment. Schaudinn (1902a) thought there was little doubt that these observers were actually dealing with the parasite of malignant tertian malaria, the name for which, according to him, becomes *P. immaculatum* Grassi and Feletti, 1892. After this, Welch (1897) proposed the name *Hæmatozoon falciparum*, so that the correct name for the parasite will be *P. falciparum* (Welch, 1897), if it be considered that Grassi and Feletti's name, *P. immaculatum*, was employed for a parasite which cannot definitely be identified with that of malignant tertian malaria. On account of this doubt, the name *P. falciparum*, now in general use, will be employed here.

PLATE XIII.

Plasmodium malariæ (1-15) AND *Plasmodium falciparum* (16-40) AS SEEN IN DRIED BLOOD-FILMS STAINED WITH LEISHMAN STAIN. (× 2,000).

Plasmodium malariæ :

1. Young ring form.
2. Young band form.
3. Slightly older parasite with granule of pigment.
4-6. Growth of schizont.
7-12. Nuclear multiplication and schizogony.
13. Older band form of nearly mature gametocyte.
14. Female gametocyte (macrogrametocyte).
15. Male gametocyte (microgametocyte).

Plasmodium falciparum :

16-24. Young ring forms.
25-26. Growth of schizont and development of pigment; these forms usually occur in the internal organs, but are occasionally seen in the peripheral blood.
27-30. Nuclear multiplication and schizogony; these forms occur rarely in the peripheral blood.
31-32. Deeply stained cells containing young ring forms, and showing Stephens' and Christopher's or Maurer's dots on the surface of the cell.
33-35. Irregular or amœboid young forms, showing tendency to fusion of one or more parasites (" *Plasmodium tenue* " of Stephens).
36-37. Developing gametocytes (crescents).
38 and 40. Female crescents (macrogametocytes) showing remains of host cell.
39. Male crescent (microgametocyte).

(ORIGINAL.)

PLATE XIII.

[To face p. 934
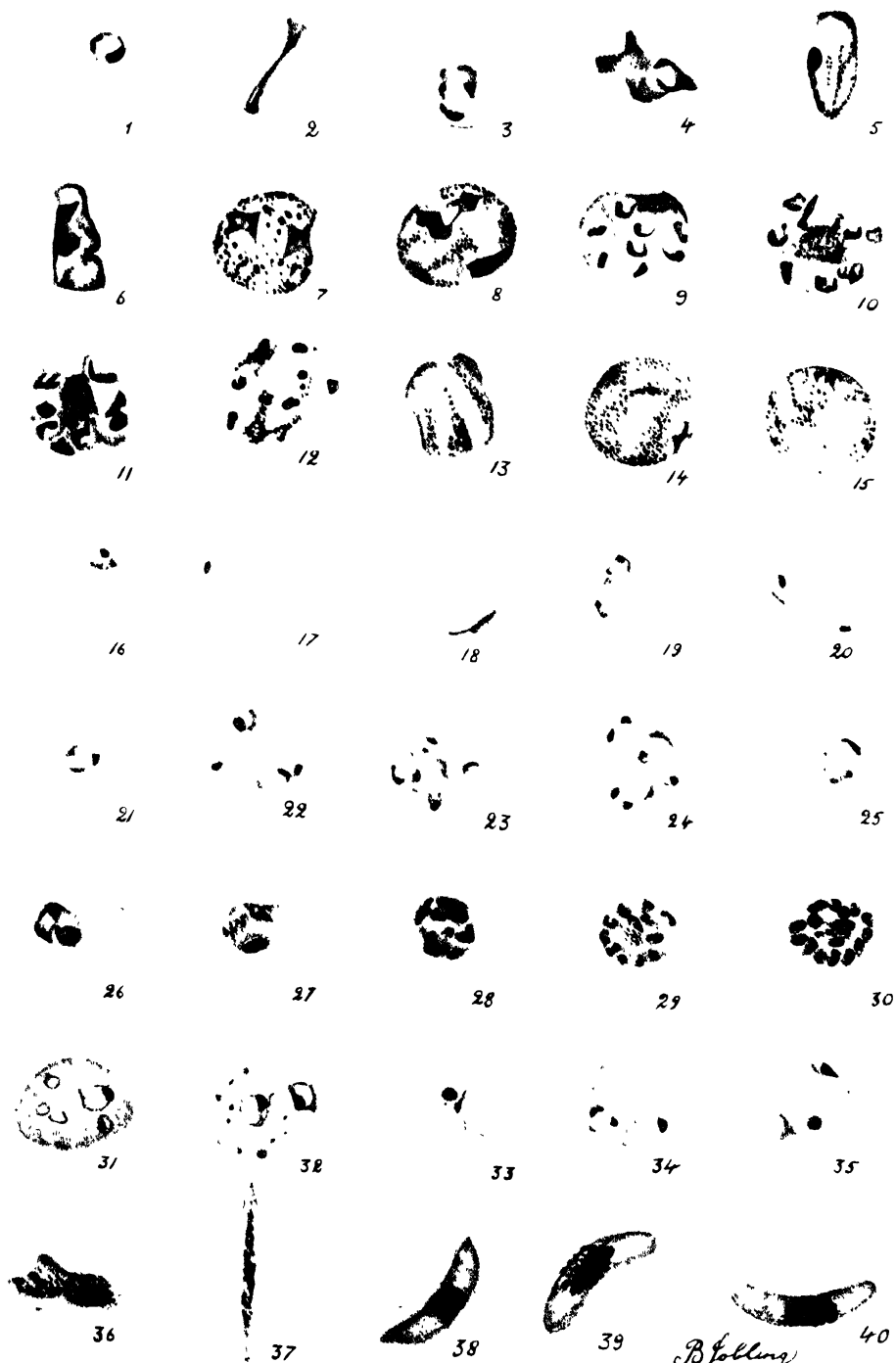
Thomson, J. D., and Woodcock (1922) have attempted to justify the inclusion of the parasite in the separate genus, *Laverania*. Their chief argument is that the gametocyte or crescent is enclosed by a capsule which determines the shape of this stage. Quite apart from the fact that it is difficult to understand how the shape can be determined by a capsule which an organism itself forms around its body, the writer is not convinced that any such capsule exists other than that produced by changes in the part of the red cell in contact with the parasite. If such a capsule exists around the female crescent, it would be highly probable that there would be similar structures around the gametocytes of *P. vivax* and *P. malariæ*, though they might be so delicate as to escape notice. The authors admit that the male crescent possesses a more delicate capsule than the female crescent. The life-cycles of these malarial parasites of men are so similar in every respect that if even a capsule did exist this would not justify a generic distinction (see p. 940).

Malignant tertian malaria of man receives its name from the fact that very heavy fatal infections are not infrequent and the attacks, though occurring much more irregularly than in the benign tertian malaria, often show a tendency to recur every alternate day.

The Cycle in Man.—The developmental cycle in man (Plate XIII., 16-40, p. 934) corresponds closely with that of *P. vivax*. There are, however, important differences, one of which is that the organism is distinctly smaller than *P. vivax*. The youngest stages arising from the penetration of merozoites are minute ring forms not more than one-sixth the diameter of the red blood-corpuscle. In heavy infections 25 per cent. of the red blood-corpuscles may be infected, while many of them contain two or three or sometimes a larger number of rings (Plate XIII., 16-25, p. 934). The cytoplasm of the ring surrounding the vacuole is very narrow, so that the ring form appears as a thin blue line with the round granule of chromatin or nucleus at one side. The chromatin granule protrudes beyond the border of the ring more than in the case of the larger rings of *P. vivax*. Though in dried films this stage is most usually seen in the ring form, occasionally more irregular or amœboid forms occur. The parasites may be any conceivable shape—round, rectangular, flame-shaped, or merely little streaks of cytoplasm with a red dot. In heavy infections, where several parasites occur in a single cell, the irregular forms may overlie one another and give the appearance of an irregular mesh-work in which several chromatin dots are found (Plate XIII., 34-35, p. 934). In fresh blood observed on the warm stage it will be noted that, as in the case of *P. vivax*, the parasites are in constant amœboid activity, so that the occasional occurrence of irregular forms in blood-films may be explained by the rapidity of drying which prevents the amœboid forms completely

retracting to the ring form. A feature of many of the rings is that two chromatin granules (nuclei) may be present, and it can be seen by examination of a number of parasites that these are produced by an elongation and division of the single one which was first present. This feature, combined with the fact that in multiple cell infections the cytoplasm of individual parasites may run into one another, has led some observers (Craig) to suggest that a conjugation of ring forms may occur in the blood. The presence of two nuclei may possibly indicate a binary fission, but the writer was unable to see any such division or any sign of conjugation after prolonged observations on the warm stage. It should be mentioned that both the bridge and flattened varieties of the marginal forms described under *P. vivax* are much more commonly seen in *P. falciparum* infections.

The growth of the ring forms into the adult schizonts is much more difficult to follow in *P. falciparum* than in *P. vivax*, for all the infected cells quickly disappear from the peripheral circulation, and are held up in the vessels of the internal organs. The reason for this is a tendency on the part of the infected cells to clump together into masses, as was clearly shown by Thomson, J. G. and D. (1913a), to occur in artificial cultures of *P. falciparum*. Working with cultures of the parasite in Macedonia, McLay (1922) further noted a tendency for the infected cells to cluster around and adhere to the large mononuclear cells in preference to any other type of cell (Fig. 401). As these cells are probably derived from the endothelial cells of the blood-vessels, there may be the same tendency in the human body for the infected cells to adhere to the walls of the capillaries of the internal organs, in which the rate of blood-flow is at a minimum. Whatever be the actual cause, the fact remains that the infected red blood-corpuscles containing the ring forms of *P. falciparum* quickly leave the peripheral circulation, so that the further development up to the stage of schizogony takes place in the internal organs. The exact stage at which the young forms leave the peripheral blood seems to vary. Sometimes it does not occur till some growth has taken place, and pigment granules have appeared in the cytoplasm. At others, it is before the pigment has formed. This fact may afford an explanation of why some observers have supposed that there exist two species of malignant tertian parasite, one which forms pigment and the other which does not. In some cases of heavy infection a certain number of the growing forms and schizonts occur in the peripheral blood, and their presence is usually an indication that the infection is an exceptionally serious and intense one.

The young ring form increases in size often by a thickening of the cytoplasm at one or two points on the ring. There appear at the same time granules of a pigment which is of a dark brown or black colour as contrasted with the light brown pigment of *P. vivax*. Further increase

in size occurs, the parasite remaining more or less compact with at most an irregular outline (Plate XIII., 25-26, p. 934). No increase in size of the red blood-corpuscles takes place, but there appear on its surface certain red-staining markings which are usually known as Maurer's dots, in spite of the fact that they were first described by Stephens and Christophers (1900, 1903a), who regarded them as of the nature of clefts in the red cell. They are larger and fewer in number than Schüffner's dots, which occur in the case of *P. vivax*, and are not so readily demonstrated.
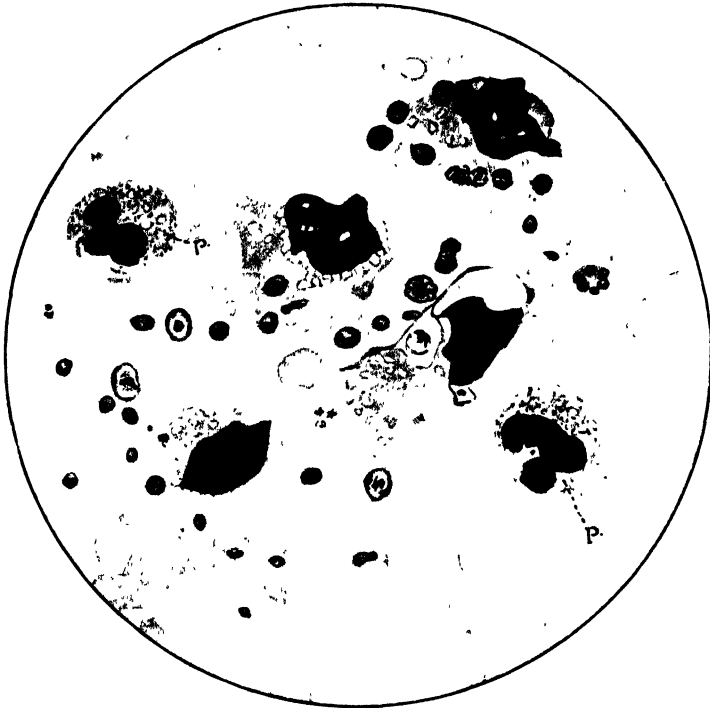
FIG. 401.—*Plasmodium falciparum* IN CULTURE, SHOWING THE TENDENCY THE INFECTED CELLS HAVE OF CONGREGATING ROUND AND ADHERING TO THE LARGE MONONUCLEAR CELLS AND NOT TO THE POLYNUCLEAR LEUCOCYTES (*p*) (× *ca.* 1,000). (AFTER McLAY, 1921.)

They are seen best in films which have been deeply stained (Plate XIII., 31-32, p. 934). Each dot is irregular in shape, and often has an angular appearance. As the parasite is approaching its full size the pigment granules, which have increased in number, run together to form a dark granular mass, which is very often situated at one side of the parasite. The nucleus multiplies by repeated divisions till eight to twenty-four are present (Plate XIII., 27-30, p. 934). The fully-formed schizont has a diameter of about 5 microns, and occupies only about two-thirds of the

diameter of the red blood-corpuscle, which is not increased in size. Very frequently, however, the shape of the cell is altered and it appears very pale in colour, as if deprived of all its hæmoglobin; at other times there is little change. The schizont breaks up into a number of merozoites corresponding with the number of nuclei. It sometimes appears that as many as thirty-two merozoites are formed from one schizont, but in these cases it is possible that the cell contained two schizonts, as sometimes happens with *P. vivax*. After schizogony, which takes place almost exclusively in the internal organs, ring forms of the parasite again appear in the peripheral circulation.

It has been mentioned above that multiple infection of red blood-corpuscles is a common feature of *P. falciparum* in its young stages. As many as six or more rings may occur in one cell (Plate XIII., 22-24, p. 934). As the parasites grow, the number of red cells with more than one parasite diminishes, so that it is probable that with growth of the parasite the cells with multiple infections break down and disintegrate. Doubly infected cells may, however, survive.

It is at the time of schizogony that the attack of malaria occurs and the temperature of the patient rises, but the attack is not so sharply defined as in the case of *P. vivax* infections, in which it commences with a definite shivering or ague attack (rigor), followed by a hot dry stage (febrile stage), which is terminated by a sweating stage when the temperature falls (Fig. 393). The fever may occur every forty-eight hours (tertian), as in *P. vivax* infections, or every twenty-four hours (quotidian). This may be due to double infections, as explained above for *P. vivax*. Some have considered, however, that distinct tertian and quotidian forms exist. In any infection the organisms are not evenly distributed through the body. In some cases the cerebral vessels are especially involved, giving rise to cerebral symptoms; in some the intestinal capillaries are most heavily infected, while in others the parasites are most numerous in the spleen or bone marrow. On this account the symptoms of malignant tertian malaria are exceedingly variable. The most serious cases are the cerebral ones, in which all types of cerebral disturbance from acute mania to coma occur, often associated with extreme elevation of temperature (hyperpyrexia). Sections of *post-mortem* material from these cases show the vessels of the brain or other organs completely blocked by aggregations of red blood-corpuscles containing the schizonts, either partially grown or in actual process of schizogony (Fig. 406). The parasites are seen, sometimes in enormous numbers, in smears, but in these the true relation to the blood-vessels is usually lost (Plate XIV., p. 956).

As in the case of *P. vivax*, certain merozoites finally grow into gametocytes, which pursue their further development in anopheline mosquitoes.

The growth into gametocytes takes place almost exclusively in the internal organs, but occasionally the young forms may be seen in the circulating blood (Plate XIII., 36-37, p. 934). The gametocytes of *P. falciparum* are characteristic in that, when fully formed, they are crescentic or sausage-shaped—hence the name crescent, which is used for them. They are about one and a half times the length and about half the breadth of a normal red blood-corpuscle. They most frequently have rounded ends, but sometimes these are pointed, giving the crescent a sickle-shaped appearance. Younger forms are slightly elongate structures, with one side convex and the other slightly concave. They gradually grow to the adult form. Sometimes the younger stages are straight elongate spindles as long as the adult forms, but much narrower, with the membrane of the enclosing red cell pushed out at each end (Plate XIII., 37, p. 934). These narrow forms increase in thickness and become curved to form the typical adult crescents. In deeply stained films the margin of the red blood-corpuscle is seen to be closely applied to the convex side of the crescent. It is stretched across the concavity where it very often shows a convexity, the corpuscle retaining some of its original shape. In some cases, especially when the film is intensely stained, the crescent appears to be completely surrounded by a somewhat irregular, red-staining border, which Thomson, J. D. (1917), has interpreted as a definite capsule, from what he believes to be its behaviour during the first stages of the subsequent development of the crescent. Two types of crescent occur—male and female gametocytes. The former consists of hyaline cytoplasm, and stains a faint blue, or often a pinkish colour, with Romanowsky stains. The nucleus is comparatively large and pale when stained, while the pigment is irregularly distributed through the cytoplasm (Plate XIII., 39, p. 934). In the female gametocyte the cytoplasm is denser and stains more deeply blue, the nucleus is compact and stains more intensely than that of the male, while the pigment tends to be aggregated around the nucleus (Plate XIII., 38-40, p. 934). In wet fixed films the nucleus of the female gametocyte is seen to be a spherical structure consisting of a nuclear membrane with a central deeply staining karyosome. The male nucleus is larger, and the chromatin is distributed through it in the form of fine granules, and not aggregated into a single karyosome.

As in the case of *P. vivax*, the number of crescents in the blood bears no relation to the number of asexual forms. Sometimes a single crescent only will be found after long search. At other times they are more numerous, and can be easily found in looking over a film. Occasionally, one or more occur in nearly every field. In such cases there may be as many as 50,000 to 150,000 in each cubic millimetre of blood. The number of gametocytes of *P. vivax* which may be present in the blood

never attains to these figures. In one important respect malignant tertian malaria differs from the benign form. In the latter, as explained above, relapses are common, and may occur at any time during several years after infection. In the former, on the other hand, relapses are less common, and the infection, as a rule, dies out more quickly, so that there is less tendency for it to persist year after year. As in the case of *P. vivax*, relapses have been ascribed, without sufficient evidence, however, to a parthenogenetic reproduction of the female gametocyte.

The Cycle in the Mosquito. — As stated above, the gametocytes of *P. falciparum* undergo their further development in anopheline mosquitoes.
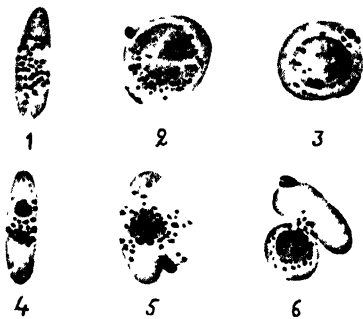
FIG. 402.—DEVELOPMENT OF THE MICRO- AND MACRO-GAMETES FROM THE GAMETOCYTES OF *Plasmodium falciparum* (× 1,500). (AFTER J. D. THOMSON, 1917.)

1. Microgametocyte (male crescent) in blood-film.
2. Spherical microgametocyte with two polar bodies.
3. Stage similar to 2.
4. Macrogametocyte (female crescent) in blood-film.
5. Cytoplasm of macrogamete escaping through opening in capsule.
6. The macrogamete has completed its escape from the cyst, at one end of which a polar body is seen.

The course of events has already been described, but certain features peculiar to *P. falciparum* will be noted here. When the crescents enter the stomach of the mosquito they retract to a spherical form, and in so doing they rupture the remains of the red blood-corpuscles, from which they escape. This process can be readily studied in a wet blood-preparation. Thomson, J. D. (1917), noted a phenomenon which had not previously been described (Fig. 402). In films which had been fixed wet with osmic acid vapour he saw appearances which seemed to indicate that the crescent was enclosed by a fairly resistant capsule. A small opening or rupture occurred at one spot, and through this the cytoplasm passed as in a hernia till the whole had escaped. Whether such a capsule is present or not it is difficult to state. The fact that in heavily stained films of blood the crescent appears to be surrounded by a deeply-staining border might suggest this. On the other hand, what has been interpreted as a capsule is more probably the remains of the red blood-corpuscle, which has become altered where it is in contact with the parasite to form a more or less definite membrane. The writer has frequently observed crescents in process of rounding off in fresh blood-preparations, but has never seen any indication that the cytoplasm normally escapes from the capsule through a small opening, as Thomson describes. It seems possible that the appearances described by him are exceptional, and

are caused by mechanical factors in the film, resulting in a rupture of the membranous portion of the red blood-corpuscle at one point only, and the escape of the cytoplasm through this opening. That part of the cell which encloses the crescent undoubtedly ruptures, owing to the contraction of the parasite to the spherical form, and this tear is usually sufficiently extensive to allow the whole organism to come out as a spherical body.

After its escape from the red blood-corpuscles, the subsequent development of the gametocytes is almost identical with that of *P. vivax*. The oökinetes, which are eventually formed, are possibly smaller than those
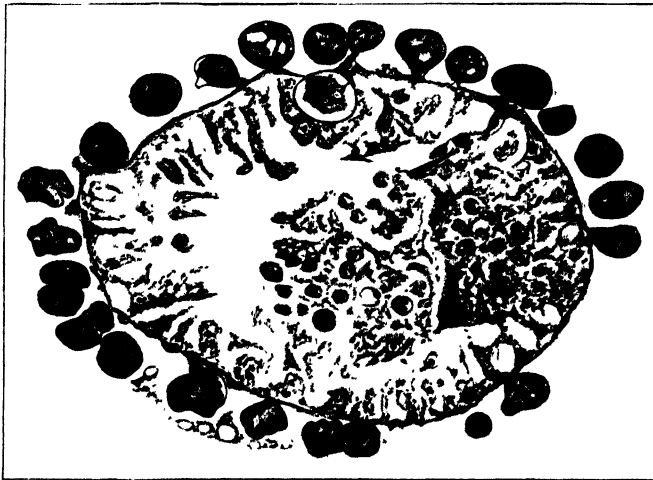
FIG. 403.—TRANSVERSE SECTION OF STOMACH OF *Anopheles superpictus* HEAVILY INFECTED WITH OÖCYSTS OF *Plasmodium falciparum* (× 200). (AFTER WENYON, 1921.)

One oöcyst is seen to be within an epithelial cell.

of *P. vivax*, and, furthermore, the dark colour of the pigment serves to distinguish them, and also the developing oöcysts, from those of *P. vivax*, which have the characteristic light brown or yellow pigment.

The fact that the number of gametocytes of *P. falciparum* which may be present in the blood reaches a figure which is never attained in the case of *P. vivax*, explains the much larger number of oöcysts of *P. falciparum* which may be found on the stomach of an anopheles (Fig. 403). In certain instances the whole stomach will be covered with oöcysts actually in contact with one another. There seems to be very little evidence that the mosquitoes are injured in any way by these heavy infections. The factors which regulate the development of *P. falciparum* in mosquitoes will be considered below (p. 958).

Plasmodium malariæ (Laveran, 1881).—This is the organism causing the disease of man known as quartan malaria from the fact that the attacks of fever occur at intervals of seventy-two hours or every fourth day of the disease (Plate XIII., 1-15, p. 934). *P. malariæ* is the parasite which was first observed by Laveran, and named by him *Occillaria malariæ*, a name which also included the other malarial parasites of man. Later, Grassi and Feletti (1892) referred to the parasite of quartan malaria as *Hæmamæba malariæ*. It was first separated from the parasite of benign tertian malaria as a distinct type by Golgi (1886).

The Cycle in Man.—The cycle of *P. malariæ* in man is very similar to that of the forms described above. The ring forms are about the same size as those of *P. vivax*, and have a diameter of about one-third of that of the red blood-corpuscle (Plate XIII., 1, p. 934). The cytoplasm, however, is generally regarded as being denser, and hence staining more deeply than that of *P. vivax*. It is very difficult, if not impossible, to distinguish the ring forms of *P. malariæ* from those of *P. vivax*. Growth takes place more slowly than in *P. vivax*, the schizont being fully formed in seventy-two instead of forty-eight hours (Plate XIII., 2-6, p. 934). During the growth period *P. malariæ* exhibits little amœboid activity, and accordingly does not show the irregularities of *P. vivax*. In this respect it resembles *P. falciparum*. The various stages do not, as a rule, show pseudopodial prolongations, though these may occur very exceptionally. After a few hours' growth pigment granules begin to appear in the cytoplasm, and these are coarse and of a dark colour, and resemble those of *P. falciparum* rather than those of *P. vivax*. As growth proceeds, the organism often shows a tendency to be stretched as a band (band form) across the diameter of the corpuscle (Plate XIII., 2, 5, 6, 13, p. 934). These band forms are very characteristic of *P. malariæ*, in which they are often seen. The bands may be quite narrow in the younger forms, or nearly as broad as long in the older ones. They occur, however, though much more rarely, in *P. vivax* and *P. falciparum*. The growth of *P. malariæ* produces very little change in the red blood-corpuscle, and there is no increase in its size. Sometimes, however, it may appear smaller than the normal cell and also, in stained films, of a deeper colour. Stippling of the cell (Schüffner's dots, Maurer's dots) does not occur in the great majority of *P. malariæ* infections. Very rarely dots resembling those seen in the case of *P. vivax* have been noted. Partially grown forms of *P. malariæ* are difficult to distinguish from partially grown stages of *P. falciparum* when the latter occur, as sometimes happens, in the peripheral blood. It should be remembered that *P. falciparum*, when fully grown, does not occupy more than two-thirds of the diameter of the cell, so that when it reaches a stage which has a diameter of about

half that of the cell, it will be nearing maturity. Its pigment granules will already be aggregating into a single mass, which is frequently situated at one side of the parasite. Furthermore, the nucleus will be showing signs of multiplication. A stage of *P. malariæ* of this size is not so near maturity, since the mature forms completely fill the cell. Accordingly, its pigment will be still scattered and its nucleus still single. As *P. malariæ* attains its full size it becomes more circular in outline, and almost completely fills the cell, while the dark granules of pigment become aggregated to form a mass which is sometimes central in position. The nucleus divides by repeated binary fission to form six to ten daughter nuclei (Plate XIII., 7-10, p. 934). The mature schizont then has a diameter of that of a normal red blood-corpuscle. The cytoplasm divides into as many merozoites as there are nuclei. If the pigment has aggregated at the centre of the parasite, the nuclei become arranged around its periphery, while the cytoplasm divides along radii to form the merozoites (Plate XIII., 11-12, p. 934). At this stage it has the appearance of a rosette (rosette form), the pigment forming the centre and the merozoites radiating from it; usually, however, the pigment is not centrally placed, and the merozoites have not the definite rosette arrangement.

In the fact that during the whole growth period the infected cells remain in the peripheral circulation *P. malariæ* resembles *P. vivax*. It is possible, however, that a larger number of parasites occur in the internal organs than in the peripheral circulation during the period between the attacks of fever. The infections with *P. malariæ* are, as a rule, less intense than those of *P. vivax*. In blood-films from ordinary cases the organisms are usually scanty. Just as in the case of *P. vivax*, double infections with *P. malariæ* may occur. In such cases one generation of parasites is twenty-four hours behind the other, and as the growth period occupies seventy-two hours, there will be attacks of fever on two successive days, followed by one day's freedom from fever. Infections with three generations are also described, in which case there will be fever every day. In the mixed infections the organisms will be found in the blood in various stages of growth, one generation always reaching maturity at the time the attack of fever is expected.

The gametocytes of *P. malariæ* are spherical bodies completely filling the red blood-corpuscles, which are of normal dimensions (Plate XIII., 13-15, p. 934). They can be distinguished as male and female, the latter staining more deeply blue and having a more compact nucleus than the former. The pigment granules are scattered irregularly through the cytoplasm. They can be distinguished from the nearly mature schizonts, which at this stage are beginning to show signs of nuclear multiplication, while the pigment is becoming aggregated into a single mass of granules.

The young gametocytes, as in the case of *P. vivax* and *P. falciparum*, are formed mostly in the internal organs. When they do occur in the peripheral circulation, they are very difficult to distinguish from partially grown schizonts.

Double infection of red cells with the ring or partially grown forms occurs, but less commonly than in *P. vivax* infections. The writer has also seen two mature gametocytes in an enlarged cell, and also a gametocyte and a schizont, producing appearances like the so-called parthenogenetic stages described by Schaudinn for *P. vivax*. The effect of *P. malariæ* on the red blood-corpuscle has been mentioned. In some films most of the infected cells appear to be of normal size, while in other cases the general impression gained in looking over a film is that a very large percentage of the parasites are in cells, which are distinctly smaller than the uninfected ones.

The Cycle in the Mosquito.—The course of development of *P. malariæ* in anopheline mosquitoes is very similar to that of *P. vivax*, from which, however, the various stages can be distinguished by the colour of the pigment. In this respect they resemble those of *P. falciparum*, which also possesses a dark brown or black pigment.

Mixed Infections with More than One Species of Malarial Parasite.

In contrast to the double or triple infections, which may occur in the case of *P. vivax* and *P. malariæ*, where two or more generations of the same species of parasite are present in the blood at one time, there occur not infrequently mixed infections of more than one species. The commonest of these combinations is to find *P. vivax* associated with *P. falciparum*, as is to be expected, since both these forms are more commonly met with than *P. malariæ*. The latter, however, is often seen in association with the other two species, and occasionally all three forms have been seen in a single blood-film. In determining these mixed infections, it is essential to see undoubted forms of each species. If rings alone were present, it would be exceedingly difficult to decide whether they all belonged to one species or to two, though the association of large rings and small rings might lead one to suspect that both *P. falciparum* and *P. vivax* were present. If, however, crescents were seen, and also older forms of *P. vivax*, in which the characteristic irregular forms, enlargement of the red cell, and Schüffner's dots were evident, there could be no doubt of the mixed nature of the infection. Naturally, the determination of a triple infection presents greater difficulties, since many stages of *P. malariæ* are with difficulty distinguished from those of *P. falciparum* when the latter occur in the peripheral blood, as they occasionally do in heavy infections.

Another important feature of mixed infections is that, though two or even three species of parasite are present, only one may be found. An individual infected with both *P. vivax* and *P. falciparum* may have an attack of malaria in which only the latter is evident, while at a later attack only *P. vivax* appears. It has frequently happened that persons become infected in malarious countries with both these forms, but that *P. falciparum* predominates to such an extent that it alone is detected. After they leave the malarious country, the *P. falciparum* infection gradually disappears. An attack of malaria occurs, and is found to be due to the more persistent *P. vivax*, which was not originally seen, and which in some way was suppressed by *P. falciparum* when it was actively multiplying. This fact has been used by the upholders of the unicist theory as proof of the transformation of one form of malarial parasite into another (see p. 953).

Differential Diagnosis of Malarial Parasites of Man.

From the above description of the malarial parasites it will be realized that certain stages can be very readily identified. Those of *P. vivax*, in which there is definite enlargement of the red blood-corpuscle, development of Schüffner's dots, and characteristic light brown pigment, are easily diagnosed. The crescents of *P. falciparum*, as also the mature form of *P. malariæ*, in which the unenlarged red cell is entirely filled by the parasite, cannot very well be mistaken for other forms. In other cases, however, there is some difficulty. The various ring stages, especially when only isolated ones can be found in a film, are sometimes impossible to determine. Partially grown forms of *P. malariæ* may not be separable from partially grown stages of *P. falciparum* when the latter, as they do exceptionally, remain in the peripheral circulation. In such doubtful cases, in order to arrive at a definite diagnosis, it may be necessary to make examinations of the blood at intervals of some hours, when the indeterminate forms will have grown into ones which are more characteristic of any particular species.

Ring Forms.--Those of *P. vivax* and *P. malariæ* have a diameter of one-third to one-half of the red cell, while those of *P. falciparum* occupy about one-sixth of the cell. In any infection irregular forms may occur, such as angular forms, amœboid forms, streak forms, flame-shaped forms, and, in fact, forms of any conceivable shape. In the case of the two first named parasites, these irregular forms are larger and composed of more cytoplasm than in the case of *P. falciparum*, since they contain the same amount of cytoplasm as the rings. The comparison is made, of course, of parasites of the same age at the time of, or immediately after, the attack of fever before any appreciable growth has occurred, and before any

pigment has appeared. The irregular forms are more commonly seen in
P. falciparum infections than in the others, and this remark applies also
to the flattened marginal forms and the bridge forms described above.
Ring forms with two chromatin dots are more common in *P. falciparum*
infections, while very heavy infections, in which many of the red cells
contain more than one parasite (multiple infection of red cells), are also
more commonly seen in the case of *P. falciparum*. The writer has,
however, seen as many as six rings of *P. vivax* in one cell. A certain number
of parasites are not in the ring form, and have a more solid appearance
from the fact that no vacuole is present. These, again, are smaller in the
case of *P. falciparum*. They represent either merozoites which have not
yet developed the vacuole, or very young gametocytes which will even-
tually leave the peripheral circulation to develop further in the internal
organs.

In many cases, however, it may be impossible to make a diagnosis
from ring forms alone. Those of *P. falciparum* may be larger than is
usually the case, while those of the other parasites may be smaller. In
the film, however, there are frequently to be found other older stages and
gametocytes. These can be more certainly identified, and their presence
makes it probable, though by no means certain, that the ring forms belong
to the same species. It is possible, however, for a large number of ring
forms of one species to be associated with gametocytes of another, in
which cases the gametocytes might produce an erroneous impression of
what the ring forms actually are.

The differentiation of the ring forms of *P. vivax* from those of
P. malariæ, when only these forms are present, is almost impossible. The
large rings are most usually *P. vivax*, because this is a much commoner
parasite than *P. malariæ*. It will appear, therefore, that very frequently
the determination of the species from the ring forms alone cannot be made
with any certainty, and the diagnosis of the ring form often depends on
the presence of other more easily recognized stages which are present at
the same time, or later at subsequent examinations when the parasites
have grown.

Partially Grown Forms.—In the case of *P. vivax*, these are recognized
on account of the infected cell being larger than the uninfected cells, and
by the presence of Schüffner's dots. Furthermore, the majority of forms
are very irregularly shaped, and they all have the light brown pigment.
Partially grown forms of *P. malariæ* are more compact, and the red cells
are neither enlarged nor possess Schüffner's dots. The pigment is dark
brown or almost black in colour. The band forms are more commonly
seen in *P. malariæ* than in the other species. Partially grown forms of
P. falciparum do not, as a rule, occur in the peripheral blood. When they

do, however, they can hardly be distinguished from partially grown forms of *P. malariæ*. When nearly full grown they are more easily identified, for they occupy not more than two-thirds of the red cell, the dark brown pigment is aggregated into one clump, often laterally placed, and the nucleus will be showing signs of multiplication. Neither in the case of *P. malariæ* nor *P. falciparum* is there any enlargement of the cell. In deeply stained films, the cells infected with *P. falciparum* may show the coarse red stippling of Maurer's dots. Partially developed gametocytes do not, as a rule, occur in the peripheral blood. If they do, those of *P. vivax* are distinguished from the partially grown schizonts by their compact form. The partially grown gametocyte of *P. malariæ* resembles the partially developed schizont, and cannot be identified with certainty. They are distinguished from those of *P. vivax* by the dark pigment and normal size of the red cell. Those of *P. falciparum* are either elongate narrow structures or slightly curved bodies which are evidently developing the crescent form. The growing schizonts of *P. malariæ* are usually rounded, but may assume other forms.

Fully Grown Forms.—The fully grown stages of *P. vivax* are recognized by the enlargement of the cell, Schüffner's dots, and the light brown colour of the pigment. The schizonts are distinguished from the gametocytes by the number of nuclei or merozoites (about sixteen), together with the light brown pigment, which is clumped in one or two masses. The mature gametocyte of *P. vivax* is distinguished from the schizont by the possession of a single nucleus and the distributed condition of the pigment. The female gametocyte stains more deeply blue, and has a small compact nucleus, while the male gametocyte stains more faintly blue or a reddish colour, and has a larger and paler nucleus. The fully grown forms of *P. malariæ* completely fill the red cell, which is of normal size. Schüffner's dots do not occur. The schizonts have about eight nuclei or merozoites, and the dark pigment is aggregated into a single dark mass. The gametocytes have single nuclei and scattered pigment. The male has a large pale nucleus and stains a pale blue or pink, while the female has a small, deeply staining, compact nucleus and deeply staining blue cytoplasm. The fully grown schizonts of *P. falciparum* do not, as a rule, occur in the peripheral blood. When they do, they are recognized by their size. They occupy only about two-thirds of the diameter of the red cell, which is not enlarged, while the pigment is aggregated into a single dark mass. The number of nuclei or merozoites is generally about sixteen. They are thus distinguished from the schizonts of *P. malariæ* by being smaller, and producing a larger number of merozoites. In deeply stained films, Maurer's dots may be present. The fully formed gametocytes are crescent-shaped and easily distinguished from all other forms. The female gameto-

cyte stains a deep blue colour, and has a deeply staining, compact central nucleus, around which the pigment granules are aggregated. The male stains a pale blue, has a larger and less deeply staining nucleus, while the pigment is scattered.

Doubtful Species or Abnormal Forms of Human Malarial Parasites.

In the above description of the three human malarial parasites, only what are to be regarded as the typical forms have been considered. As already explained, in many cases without any treatment the infections naturally abate as the result of some protective factor in the body. It is probable that some substance is produced which has a deleterious action on the parasite. A more rapid suppression of an infection is brought about by the administration of quinine. With these factors, and probably others of which there is at present no knowledge, acting upon the parasites, it is not to be wondered at that occasionally forms are seen which do not correspond in every way with the typical ones. Very commonly what are undoubtedly degenerating or damaged forms are seen after quinine treatment. The altered parasites may still grow and reproduce by schizogony. In the case of *P. vivax*, ragged-looking schizonts sometimes appear. They may be smaller and contain fewer nuclei than the normal schizonts, while the irregular staining indicates some degenerative process. In other cases, the departure from what may be considered the normal type is less marked, and consists of an unusual shape of the parasite, increase or diminution in the size of the nucleus or its chromatin element, unusual behaviour of the infected red cell, and other changes. It has to be remembered that the malarial parasites are almost invariably studied in dried films stained by Romanowsky stains. In many respects these stains, though giving very beautiful pictures, are very unreliable, for they are subject to variations, which cause them to stain differently at different times. Thus, in the case of typical *P. vivax*, the absence of Schüffner's dots, which is sometimes noted, is almost invariably due to a poorly acting stain. The intensity of staining, depending on the quality of the stain, or the length of time it has been allowed to act, produces remarkable variations in the appearance of the parasites and the infected cells. When films are deeply stained, many more granules take a red coloration than when they are lightly stained, and it is certainly incorrect to regard all red-staining granules as chromatin. It has accordingly happened that new species of human malarial parasites have been described from time to time. Generally these have been seen in single or very few blood-films taken from a case on one or two occasions. It has been impossible to follow the cycle of the parasite, as has been done in the case of the three

well-established species. Until this can be done, and the parasite has been proved to retain its characters, both in human and mosquito passages, it is quite unjustifiable to introduce specific names. Laveran (1914) remarked that he had long held that the descriptions which were usually given of the species of malarial parasites were too schematic, and that in practice intermediate and veritable transition forms were frequently met with. This remark is certainly correct, and if every slight variation from the normal is considered of specific importance, there is no limit to the number of species which might be created. Vialatte (1922) goes even further, and concludes that, owing to the fact that every grade of transition between the three types of malarial parasite exists, it is at present impossible to regard the malarial parasite as representing more than one species.

The following forms, which have been given specific names, are mentioned here, but it seems to the writer more logical to regard them all as unusual forms of one of the three well-known species. Catanei (1923), in an exhaustive review of the subject, comes to a similar conclusion.

Plasmodium vivax var. **minuta** Emin, 1914.--Emin (1914) described, under the name of *P. vivax* var. *minuta*, a malarial parasite which he had found in the blood of pilgrims in Camaran Island in the Red Sea. Films were studied by Ziemann (1915), who considered it a distinct species, to which he gave the name *P. camarense*. The features which distinguish it from the three well-known species are, according to Emin:

1. Marked amœboid activity, so that the parasites are very irregularly shaped.

2. The red blood-corpuscle is neither enlarged nor altered in colour.

3. Schüffner's dots are well marked, and may be in the form of filaments.

4. Nuclear multiplication commences when the parasite is only half the size of the red cell.

5. The schizont is only two-thirds the size of the red cell. It occurs in the peripheral blood, and four to ten merozoites are produced.

6. Very little pigment is produced, and the whole of it remains in a residual body at schizogony.

7. The gametocytes are round, and reach a size of three-quarters of the red cell.

Ziemann pointed out that even in the very young forms the chromatin is well marked, and often divides into two or three masses, as commonly occurs in *P. falciparum*. The dots on the red cells are, according to Ziemann, more like Maurer's dots than Schüffner's dots.

Stephens (1922) has described as *P. ovale* a parasite which, he says, appears to resemble that described by Emin. In all the stages of its

growth it resembled *P. malariæ*, but differed in the absence of the band forms so commonly seen in infections with this parasite. Furthermore, the infected corpuscles in many cases appeared oval in outline, were slightly enlarged, paled, and stippled with Schüffner's dots. The cycle of development apparently occupied twenty-four hours. It will be noted that in Emin's parasite the growing forms were very irregular in shape. In Stephens's form the parasites were always compact (Fig. 404). Stephens evidently thinks it possible that the two forms are identical, but states that he was unable to decide this point, as he could not obtain preparations of Emin's parasite. Nevertheless, he named it *P. ovale*, overlooking the
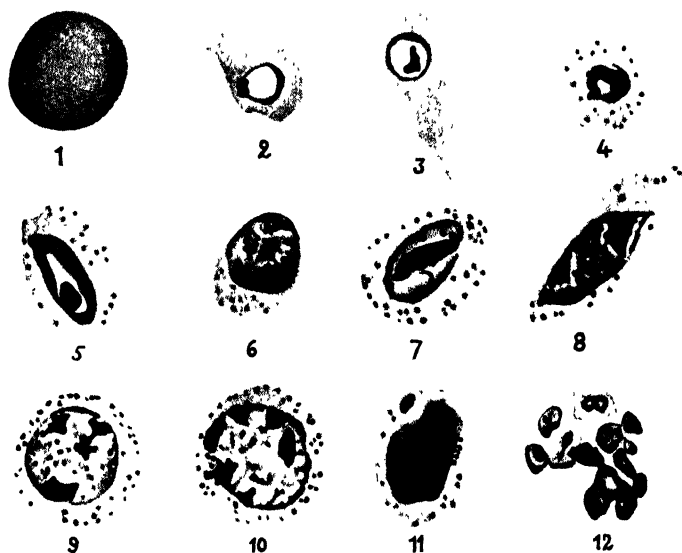
FIG. 404.—*Plasmodium ovale* (× 1,800). (AFTER STEPHENS, 1922.)

1. Normal red blood-corpuscle.   2-3. Ring forms.
4-7. Partially grown forms.   8-12. Nuclear multiplication and schizogony.

fact that Ziemann (1915) had already proposed the name *P. camarense*. It is worthy of note that eight days before the appearance in the blood of the forms regarded as a new species, the blood-films made from the case showed typical forms of *P. vivax*.

Forms similar to this one have been seen by the writer in Mesopotamia and Macedonia, and he is inclined to regard them as aberrant types of *P vivax* or *P. malariæ* (Plate XII., 31-35, p. 926). Knowles (1923, 1923*a*) has expressed a similar opinion. If, however, the parasite is a true species, of which there is insufficient evidence, the correct name will be *P. minutum* Emin, 1914.

Plasmodium perniciosum Ziemann, 1915.—Ziemann believes that the parasite of malignant tertian malaria in West Africa is distinguishable from that of Italy and other countries where the true *P. falciparum* occurs. *P. perniciosum* is said to differ from *P. falciparum* in the following characters:

1. Small quantity of pigment.
2. Darker colour of the pigment.
3. Complete disappearance from the peripheral blood after the rings have been formed.
4. Want of " brassy tint " of infected red cells.
5. Schizonts occupy only one-third to one-half the diameter of the cell.
6. The number of merozoites is only twelve to sixteen.
7. Crescents are not produced so abundantly, and they are smaller and plumper.

The writer, who has studied both the European and the West African types, has frequently seen the African form behave in a manner the reverse of that which is supposed to be characteristic of it by Ziemann. The parasites may or may not completely disappear from the peripheral blood, the crescents may be produced in enormous numbers, and their shape is subject to distinct variations; the pigment in the schizonts is often large in quantity, so that there seems little reason for regarding *P. perniciosum* as a distinct species.

Plasmodium tenue Stephens, 1914.—This is the name given to a malarial parasite seen by Stephens in a single blood-film sent him from India (Fig. 405). Only young forms were present, and they were characterized by their marked amœboid form, and by the large size of the nuclear chromatin and its irregularity in shape. Balfour and the writer (1914) pointed out that such forms were by no means uncommon in *P. falciparum* infections, and that there was no reason to regard *P. tenue* as a distinct species (Plate XIII., 34-35, p. 934). Knowles (1923a) expresses a similar opinion. Sinton (1922b) in India has given an account of a case of malaria in which these forms appeared regularly, and he is inclined to regard Stephens's view as correct. It would appear, however, that the name *P. tenue* cannot be employed, as Laveran and Marullaz (1914) used the name *Hæmamœba tenuis* for a small form of the bird malarial parasite which belongs to the genus *Plasmodium*. Sinton thinks it possible that *P. tenue* is identical with the parasite described by Grassi and Feletti (1892) as *Hæmamœba immaculata* (see p. 934).

Chalmers and Archibald (1920) described what they term the " tenue " phase of *P. vivax*. It occurred in an undoubted infection with *P. vivax*, and showed a departure from the usual picture in that many of the red

cells contained several rings instead of the more usual one or two (Plate XII., 28-29, p. 926). As a result of the multiple infection and the amœboid activity, the rings had run into one another or overlapped, so that the appearance of a reticulum, with several chromatin dots, was produced. The interpretation of this phenomenon given by these observers, that it represents an attempt at a primitive method of binary or multiple fission at this stage of development, is highly speculative. It is more probable that the reticulum was the result of fusion or apparent fusion, as the result of overlapping, of several actively amœboid forms. The writer has seen on
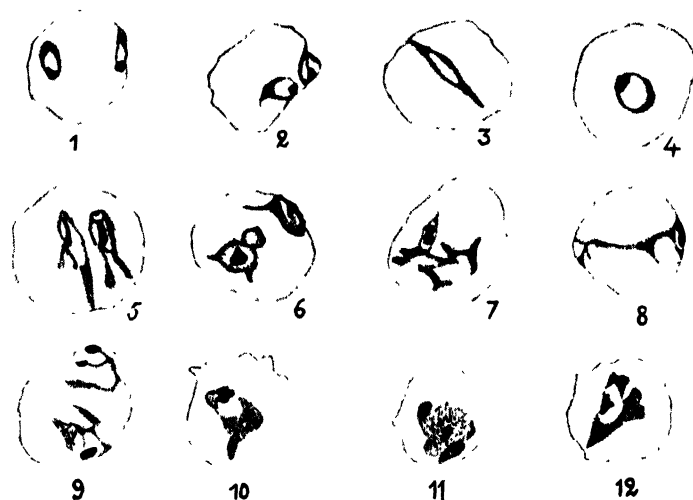
FIG. 405.-- *Plasmodium tenue* STEPHENS, 1914 (× *ca.* 2,000). (AFTER SINTON, 1922.)

1-2. Young forms probably varying in age from three to eight hours.
3-6. Medium-sized, irregular, or oval-tailed forms, probably varying in age from twelve to twenty-four hours.
7-9. Forms during "tenue" phase, probably varying in age from twenty-eight to thirty hours.
10-12. Large oval or polyhedral forms, probably varying in age from thirty-six to forty hours.

several occasions large infections of *P. vivax*, in which many cells showed two or more young forms. On one occasion as many as six occurred in one cell.

The occurrence of pigmented and unpigmented varieties of *P. falciparum* has been explained above by the fact that the young forms disappear from the peripheral circulation either before or after pigment has appeared in their cytoplasm. It seems not improbable that the time of disappearance of the growing forms of *P. falciparum* from the peripheral blood varies considerably. Very frequently the only forms found are the young minute ring forms which disappear into the internal organs after a few hours. In other cases the disappearance is delayed, and older forms

with one or two granules of pigment may be found. A further delay of twenty-four hours or more will lead to the discovery in the films of still larger parasites, definitely pigmented, and sometimes markedly amœboid in form, the larger forms retaining their irregularity during the drying process of film-making more easily than the younger ones, which contract to the definite ring more quickly.

Many attempts have been made to discover a particular type of parasite, which may be supposed to produce blackwater fever, which occurs chiefly as a complication of *P. falciparum* infections. Wright (1920) described in great detail with many coloured plates the parasites he had found in the blood in association with this disease in India. He regarded some of the forms as *P. falciparum,* and others, on account of their shape, as piroplasmata, or a new species of malarial parasite. There is no doubt that all the forms described are either *P. falciparum* or *P. vivax. P. caucasicum,* described by Marzinowsky (1916), is in all probability a form of *P. falciparum,* from which it is said to differ in minute details only.

It cannot be too forcibly emphasized that the parasites of malaria, like all other living organisms, are liable to appear occasionally in abnormal form, and that more evidence than the mere casual appearance of these is required before new species are created.

Theory of Unity of the Malarial Parasites.

It should be mentioned here that Laveran and other observers have maintained that the three malarial parasites of man belong to one species, and that the various appearances assumed by it and interpreted as due to a distinction of species are the result of temporary alterations in structure brought about by certain influences not properly understood. Under certain conditions the form of *P. vivax* is assumed, while at others that of *P. falciparum.* There seems no real ground for this hypothesis, which is not upheld by the great majority of those who have studied malaria. Grassi (1919), in direct opposition to his previous views, expressed it as his belief that only a single species exists, and that the form known as *P. falciparum* relapses in Italy in March and April as *P. vivax.* The same opinion is expressed by Grassi and Sella (1920). Vialatte (1922) has arrived at the same conclusion. It has been proved, however, that direct inoculation of infected blood gives rise to infections with the type of parasite inoculated. Thus, Mühlens and Kirschbaum (1921) inoculated human beings with malaria with the object of testing its effect on general paralysis. They passed *P. vivax* in series through twenty cases, *P. falciparum* through four, and *P. malariæ* through three. In all cases the parasites retained their original characters. Bastianelli and Bignami (1899),

in transmitting malaria from man to man by means of *Anopheles maculipennis*, found the type inoculated corresponded with the one taken up by the mosquito. Similarly, Bruce Mayne (1920), working in America, has shown that mosquitoes convey only the form with which they were originally infected. Mühlens, Weygandt and Kirschbaum (1920) inoculated both *P. falciparum* and *P. vivax* from man to man both during the winter and summer in Germany. In no case did the parasites which appeared in the inoculated individuals depart from the type introduced. In a later paper Mühlens and Kirschbaum (1924) have given further evidence of the constancy of type. In a long series of passages, which in the case of *P. vivax* has reached forty, the three species have maintained their characters throughout, and shown no tendency to change from the one to the other. Yorke and Macfie (1924a) have inoculated seventy cases of general paralysis involving twenty-three passages with *P. vivax*, and have infected forty-one cases with the same parasite by the bites of *Anopheles maculipennis*, and have noted no change in the morphology of the parasite.

Possibility of Animal Reservoir—Susceptibility of Man and Animals to Inoculation.

The fact that persons sometimes contract malaria after visiting certain mosquito infected districts which appear to be uninhabited by man has raised the question of an animal reservoir for the human malarial parasites. A large number of animals of different species have been examined, with the result that a number of malarial organisms have been discovered in them. These are, however, distinct from the human forms, for their inoculation into man has not been followed by infection, nor has inoculation of the human parasites into animals, as carried out by many observers, been followed by infection, except in one instance. Bass (1922), who points out that horses, mules, dogs, foxes, monkeys, rabbits, mice, guinea-pigs, hedgehogs, bats, wolves, cats, pigeons. doves, magpies, screech-owls, turtles, frogs, and lizards have been inoculated with human blood containing malarial parasites without infections resulting, has himself attempted to infect four guinea-pigs, five rabbits, and one *Macacus rhesus* with *P. falciparum*. As a control, culture was made with the same blood in human blood medium and in a medium of the blood of the animal inoculated. The parasites developed in the human blood medium, but not in the other. None of the animals became infected, though some parasites remained in the blood of a guinea-pig for twenty-four hours. In the case of a chimpanzee, Mesnil and Roubaud (1920) succeeded in producing a mild infection with *P. vivax* by inoculating infected human blood intravenously. The infection appeared in ten days, and lasted for

a similar period. Another chimpanzee inoculated in a similar manner did not become infected. *Anopheles maculipennis* infected with *P. falciparum* failed to infect a chimpanzee on which it was allowed to feed.

Reichenow (1917, 1920c) discovered malarial parasites in anthropoid apes in West Africa (Plate XV., 1-7, p. 970). The forms described resembled the three well-defined types of the human parasite. He produced some evidence that it was only those apes which were kept in association with man that harboured the parasites, and it seemed to him probable that they had actually become infected from human beings. Blacklock and Adler (1922, 1924), who studied the parasites of the chimpanzee, failed to infect human beings by inoculating them with blood of an infected ape, and also failed to infect a young chimpanzee with *P. falciparum* (see p. 970).

Inoculation of infected human blood into human beings produces infections quite readily. An instance is known to the writer in which a series of men was infected during salvarsan injections. The apparatus consisted of a receptacle connected with a rubber tube, to which the needle to be inserted into the vein was attached. A short distance above the needle a piece of glass tubing formed a window, and at each injection, by lowering the receptacle, blood was allowed to pass the window to ensure the fact that the needle was in the vein. The receptacle was then raised, and the required amount of drug run in. A sterile needle was used for each man, but the window was not changed, so that it was possible for the blood of each man to be contaminated by the blood adhering to the window from the previous injections. In this way the blood of one man, who was later found to harbour crescents in his blood, infected the ten succeeding men, and produced in them attacks of malignant tertian malaria, which were fatal in some of the cases.

Pathology of Malarial Infections.

The most striking macroscopic change in the body, due to the presence of malarial parasites, is a marked enlargement of the spleen, which in some cases reaches enormous dimensions. In acute cases it is soft and dark red in colour, while in chronic infections it is harder and may have a dark steely grey tint owing to deposition of pigment. The liver is also enlarged, but to a less extent. In countries in which malaria is endemic, the percentage of children showing enlargement of the spleen (spleen index) affords a reliable guide to the intensity of malaria in the community. Very definite changes occur in the blood. It becomes thin and watery, and the number of red cells is reduced beyond the degree that

would be expected from the intensity of any infection. That is to say, there appears to be a destruction of red cells in excess of those actually infected. The surviving red cells often show irregularities of size and shape, and contain less hæmoglobin than is normally the case. Nucleated red cells are also not infrequently seen. The actual number of leucocytes is below the normal (leucopænia), while the percentage of the large mononuclear cells, which are probably of endothelial origin, is increased. Actually, during a malarial attack, the polynuclears may be present in a percentage which is greater than normal, but between the attacks their percentage diminishes. On this account an increase in the percentage of large mononuclear cells is in favour of any condition being caused by a malarial infection. In sections of tissues from malarial subjects, the most striking feature is the pigment which has been liberated from the parasites at the time of their schizogony. It is discharged into the plasma, where it may be seen in the form of granules at the time of an attack of fever. It is, however, quickly taken up by the endothelial cells of the vessels, and also by the circulating large mononuclear cells, or even the polynuclears. The presence of pigmented leucocytes, as these circulating cells which have ingested pigment are called, is another indication that malarial parasites are present in the body. If the organs, especially the spleen from a case which has died shortly after an attack of fever, are examined in sections, the vessels will show pigment in the form of fine granules in their lumens, either free or in leucocytes, and also within the endothelial cells lining the vessels. In cases of chronic malaria, it appears that the pigment taken up by the endothelial cells at the time of an attack has been passed into the interstitial tissue, where it occurs in cells, which may again be of endothelial origin, in the form of dark brown or blackish aggregations produced by the finer granules first deposited having run together. The cytoplasm of the glandular cells of the liver and other organs may show fine pigment granules of a yellow colour. These are of a different nature from those just mentioned, and give the microchemical test for iron. They are not elaborated by the parasite, but are formed by the tissue cells from the hæmoglobin which is liberated from the destroyed red cells. They are not characteristic of malaria, but occur in other diseases associated with blood destruction (see p. 882).

In cases that have died of malaria, the capillaries of the internal organs show varying numbers of red cells infected with malarial parasites. In pernicious cases of *P. falciparum* infection the capillaries of the brain, spleen, intestine, liver, or any other organ may be completely blocked by veritable emboli of infected cells. The capillaries, as seen in smears or sections, stand out clearly owing to the clumps of pigment in the parasites (Fig. 406).

PLATE XIV
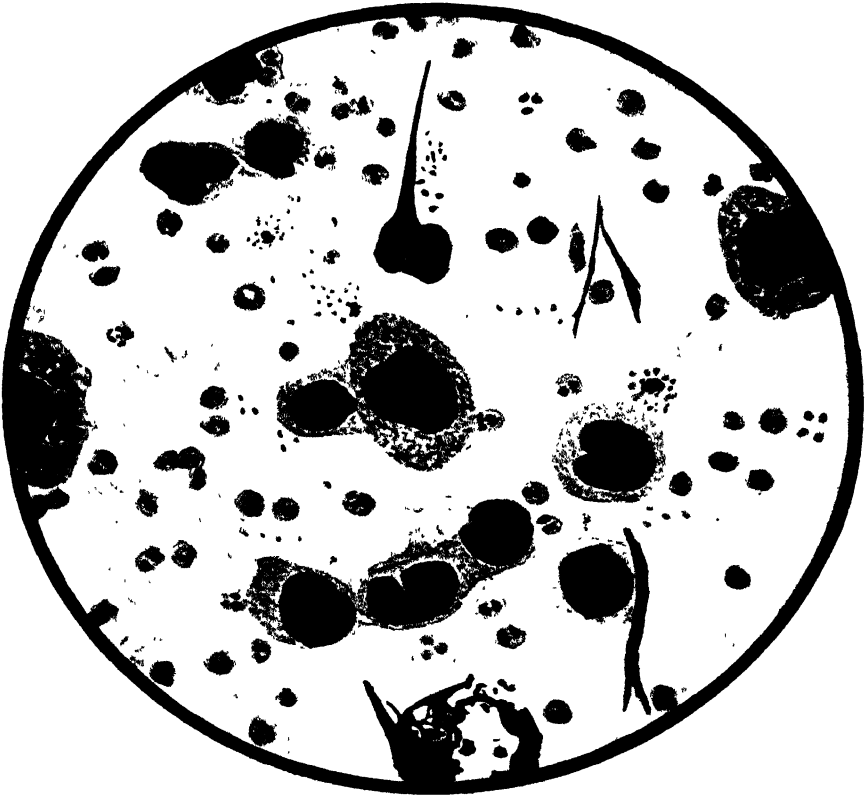
Plasmodium falciparum as seen in spleen smear of a fatal case of malignant tertian malaria (x 1,000). The majority of the parasites are fully grown schizonts each with single nucleus and pigment granules aggregated into a clump. A few parasites have two or more nuclei, while two are producing merozoites. A number of scattered merozoites occur, and some of these have invaded the red blood corpuscles. Three large endothelial cells are seen. One of them contains masses of pigment, and another two phagocyted parasites.

(Original.)

In the spleen, especially, large endothelial cells still in the vessel wall may be seen to contain quantities of pigment, infected red blood-corpuscles, free parasites, and even leucocytes which they have phago-cyted (Plate XIV., p. 956).
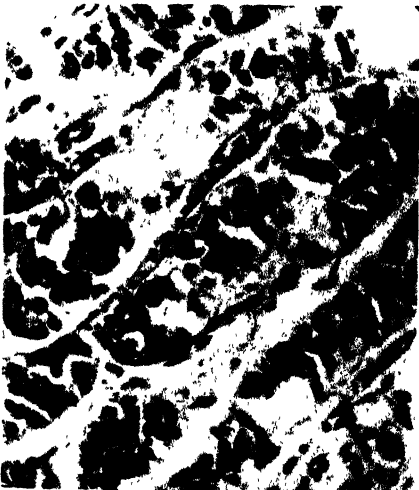
FIG. 406.—DISTRIBUTION OF *Plasmodium falciparum* IN THE CAPILLARIES OF THE INTERNAL ORGANS OF FATAL CASES OF MALIGNANT TERTIAN MALARIA ($\times$ 220). (MICROPHOTOGRAPHS BY DR. A. C. STEVENSON.)

The mass of dark pigment in each intracorpuscular parasite is clearly seen.
A. Smear of brain substance showing network of capillaries.   B. Section of brain.
C. Section of intestinal mucosa.   D. Section of pancreas.

Factors which Influence the Development and Survival of Malarial Parasites in Mosquitoes.

It has already been stated that the human malarial parasites undergo development only in anopheline mosquitoes. All attempts to infect other forms (*Culex*, *Aëdes*, etc.) have failed, and it is quite clear that the anopheline mosquitoes, as first pointed out by Grassi, are alone responsible for the spread of malaria. The mosquitoes can only infect themselves from cases in which gametocytes occur in the blood, and the number of oöcysts which develop is dependent on the number of gametocytes present. The largest infections in mosquitoes are seen in the case of *P. falciparum*, for in man this malarial parasite produces the largest number of gametocytes.

Mitzmain (1917*a*) showed that only a small percentage of gametocytes of *P. falciparum* actually develop in the mosquito's stomach, for large numbers are passed through the intestine and escape in the fæces. It is possible that in order to develop they must have reached some hitherto undetected condition of maturity in the human blood. Furthermore, Mitzmain, working with *Anopheles punctipennis*, found that of sixteen mosquitoes which had a single feed on blood containing crescents, only one became infected, while of thirty-six which fed a varying number of times, thirteen became infected. The chances of a mosquito becoming infected thus increase with the number of feeds on infected blood.

The actual development in the mosquito is directly dependent upon temperature. The first persons to study the subject were Grassi, Bignami, and Bastianelli (1899*a*). They noted that neither *P. vivax* nor *P. falciparum* would develop in mosquitoes kept at a temperature of 15·5° to 17·5° C., and Grassi (1900) considered that this was due to the prevention of microgamete formation and fertilization. At 17° C. the gametocyte of *P. falciparum* rarely flagellates, whereas at 18° to 20° C. it does so readily. The minimum temperatures for continuous development to take place were 16·5° C. for *P. malariæ*, 17·5° C. for *P. vivax*, and 18° C. for *P. falciparum*. If the temperature is sufficiently high for fertilization to take place, then, according to Grassi, development may proceed at lower temperatures. It was found that at a temperature of 30° C. *P. falciparum* would complete its development in seven days, while at 20° to 22° C. it required about twenty days. Jancsó (1904) published accounts of experiments from which he drew conclusions at variance with those of Grassi. He found that, if immediately after biting, mosquitoes were kept for twenty-four hours at 11° to 13° C., and then at a temperature of 20° to 30° C., development would take place. If, however, they remained at the lower temperature, no infection occurred. He concluded that the

low temperature did not hinder fertilization, but prevented the oökinete from penetrating the stomach wall and becoming an oöcyst.   Mitzmain (1917) recorded a series of experiments in which mosquitoes were fed on cases of *P. falciparum* infection.   After being kept for a few hours in the room with the patient, they were transferred to a living room for ten to thirteen days at a temperature of 20° to 26° C.   They were then gradually taken to outside conditions in open-air cages during November, December, and January, with temperatures of from 14·5° to 24° C., 11·5° to 20·5° C., and 14·9° to 21° C. respectively.   The mosquitoes were examined from ten to seventy days after feeding.   Of fifty-four specimens of *Anopheles punctipennis* thus treated, fifteen showed infection with oöcysts, but in no case were sporozoites found in the salivary glands.   In many cases the cysts were seen to be ruptured.   It is concluded that intermittent low temperatures restrict or prevent development, even when a favourable temperature existed during the early stages.   Eight mosquitoes which had been exposed to the intermittent low temperature were at the end of the experiment transferred to a favourable temperature (mean 24·6° C.). No salivary gland infection took place.   King (1917) showed that if *P. vivax* were allowed to develop in *Anopheles quadrimaculatus* for seven to twenty-three days at a favourable temperature. the oöcysts which had partially developed were able to withstand exposure to lower tempera- tures (− 1° C. for two days, − 0·5° C. for four days, 7·3° to 10·6° C. for six to seven days, 3·3° to 14° C. for seventeen days), and to continue their development when subsequently removed to a favourable temperature. *P. falciparum* under similar conditions survived an exposure to 1·7° to 14° C. for one to two days.   Exposures to low temperatures for longer periods resulted in degeneration of the oöcysts.   Some similar experiments were conducted by the writer (1921c) in Macedonia.   It was observed that in nature partially developed oöcysts could be found in hibernating *A. superpictus* all through the winter.   The question was raised as to the possibility of these oöcysts being able to continue their development when the temperature became more favourable in the spring.   Mosquitoes were fed on cases of *P. falciparum* infection, and partial development allowed to take place at a temperature of 22° C.   The development was completely arrested for fifteen days by placing the mosquitoes at a tem- perature corresponding to their winter hibernation (9·7° to 18·7° C.). Subsequent exposure to favourable temperature (21° to 24° C.) was followed by development of the oöcysts to maturity.   Twelve hours' exposure to a temperature of 5·5° C. did not injure the cysts.   It is thus possible that the immature cysts found under natural conditions in winter are able to develop to maturity when the temperature becomes higher, and that anopheline mosquitoes can in this way carry infection through the winter.

As regards the times necessary for the completion of development in the mosquito, it is found that *P. vivax* develops more quickly than *P. falciparum*. Thus, in *A. maculipennis* at a mean temperature of 17° to 20° C., according to Roubaud (1918), *P. vivax* requires fifteen days and *P. falciparum* twenty days for complete development, while at temperatures of about 25° C. *P. vivax* develops in eleven days and *P. falciparum* in fourteen days. In the writer's experience in Macedonia, at a temperature of 18° to 25° C. *P. vivax* completed its development in *A. maculipennis* in seventeen days, while with *P. falciparum* the oöcysts were not quite mature in the same period. In *A. superpictus*, *P. vivax* completed its development in fifteen days at a temperature of 21° to 29·7° C., while *P. falciparum* at a lower temperature of 21·5° to 23·7° C. required seventeen days. It thus appears that the development of *P. vivax* is slightly more rapid than that of *P. falciparum*, and that the difference is more marked at lower temperatures. Jancsó (1904) estimated that at a temperature of 15° to 17° C., which is below the limit at which *P. falciparum* will develop, *P. vivax* would require fifty-three days to complete its development. Roubaud (1918) sees in these data an explanation of the fact that in certain countries *P. vivax* infections occur earlier in the year than those of *P. falciparum*. As regards the upper limits at which development will take place, this depends entirely on the temperature at which mosquitoes will survive. Grassi (1900) states that *P. vivax* and *P. falciparum* complete their development in *A. maculipennis* in eight days when kept at a constant temperature of 28° to 30° C. At a constant temperature of 30° C., seven days is sufficient. The rate of development of *P. vivax* and *P. falciparum* in anopheles is approximately the same at high temperatures (30° C.), and sporozoites are found in about seven days. As the temperature falls, the rate of development of *P. falciparum* falls off more quickly than that of *P. vivax*. At a temperature of 18° to 25° C., *P. vivax* requires about fifteen to seventeen days and *P. falciparum* about nineteen days. At lower temperatures *P. vivax* still continues to develop, though *P. falciparum* has ceased to do so.

As regards the effects of the humidity of the atmosphere on the active development in mosquitoes, there is no evidence that this factor plays any part. Provided there is sufficient moisture in the air to enable the mosquitoes to live, the malarial parasites will develop normally. Temperature is a much more important factor than humidity. As Gill (1921) has pointed out, the spread of malaria may, however, be affected by lack of humidity, because the mosquitoes which hatch and ingest parasites may not live long enough for sporozoites to appear in the salivary glands.

The writer (1921c) noted in Macedonia that anopheles could be infected with *P. falciparum* from patients who were taking quinine, but

that under these conditions *P. vivax* rarely developed.   Quinine, therefore, affects the gametocytes of *P. vivax*, but not those of *P. falciparum*.   This observation accords with the results of Bruce Mayne (1920) in America. Bastianelli and Bignami (1900) and Gualdi and Martirano (1901) had previously noted that quinine taken by a patient did not prevent the crescents developing in mosquitoes, while Schoo (1902) failed to obtain development of the gametocytes of *P. vivax* under similar conditions.

It is evident that with a limited number of sporozoites in the salivary glands of a mosquito continued passage of these in the salivary fluid will lead eventually to disinfection of the mosquito.   To bring this about a number of feeds may be necessary.   Mitzmain (1916*a*) attempted to determine the number of persons which could be infected by a single anopheles.   His results appear to indicate that in a period of fourteen days as many as nine individuals can be infected with *P. vivax* by one mosquito.

As regards the duration of life of sporozoites in the salivary glands of mosquitoes, some data have been obtained.   Martirano (1902) noted that in Italy mosquitoes with infected salivary glands could not be found during the winter, and concluded that the infection was quickly lost.   Schoo (1903) kept infected mosquitoes fed only on water through the winter (October to March), and could find no sporozoites when they were examined in the spring.   Roubaud (1918) found that only degenerate sporozoites occurred in the salivary glands of a specimen of *A. maculipennis* dissected four months after feeding on a crescent case.   Cardamatis (1919) stated that he had found degenerate oöcysts in anophelines during winter in Greece, while Grassi and Sella (1920) observed sporozoites in the salivary glands of hibernating *A. maculipennis* in Italy in the winter, but in the spring no such infections were noted.   It was concluded that the sporozoites could not survive in the salivary glands through the winter. Bruce Mayne (1922) has given an account of more accurate investigations conducted in America.   He has previously (1920) shown that what were apparently dead sporozoites were found in the salivary glands of an *A. punctipennis* 158 days after feeding on a case of *P. vivax* infection. In his later experiments he demonstrated by actual feeding on volunteers that infective sporozoites were present in the salivary glands fifty-five days after *A. punctipennis* had fed on a crescent case.   Though this mosquito had produced an infection on the fifty-fifth day, it failed to infect another volunteer on the sixty-seventh day.   It was dissected on the sixty-eighth day, when typical active sporozoites were found.   In another experiment four *A. punctipennis* were induced to bite a volunteer on the seventy-fourth day.   No infection resulted, though sluggish sporozoites were present in the salivary glands of two of them when dissected some time after.   Two other mosquitoes failed to infect volunteers

on the sixty-first and sixty-sixth days, though active sporozoites were present when dissected nine and five days respectively after feeding. Sporozoites which were evidently dead were seen as late as the ninety-fifth day in the case of *P. falciparum*, and the 105th day in the case of *P. vivax*. James (1926) has shown that a batch of *A. maculipennis* infective on August 17 was still infective on November 17, though it had twenty-four previous opportunities of injecting sporozoites. During eighty-six days the mosquitoes were kept at 38° to 40° C., being transferred from time to time to a temperature of 24·4° C. One of the mosquitoes dissected on November 12 had zygotes on the stomach and sporozoites in the glands.

Barzilai-Vivaldi and Kauders (1924), working in Germany, attempted to transmit malaria to six healthy subjects by means of 120 *A. maculipennis* which had been fed on patients harbouring malarial parasites (*P. vivax*) which had been directly inoculated from man to man over a long period. No infection resulted, and dissection of the mosquitoes showed that no development of the parasites had taken place. It is concluded that after a number of direct passages the parasite eventually ceases to produce gametocytes capable of developing in mosquitoes. Such statements require very careful confirmation before they can be accepted, but they may be compared with those made by Duke (p. 512), who found that after many direct passages trypanosomes appear to lose their power of developing in tsetse flies.

Anopheline Carriers of Malaria.

It has been abundantly demonstrated that the human malarial parasites will not develop in any but anopheline mosquitoes. In any particular malarial locality all species of anopheles are not equally responsible for the spread of the disease. Naturally, those which come into most intimate contact with human beings are most often infected. In some cases anophelines are easily infected, practically every one feeding upon a suitable case developing oöcysts which reach maturity, while in others, as in the well-known instance of *A. rossii* (*A. subpictus*) of India, infection does not occur, or only very rarely. Thus at Mian Mir in the Punjab, Stephens and Christophers (1902) dissected 259 *A. culicifacies* and 496 *A. rossii*, with the result that 12 of the former and none of the latter were found infected. At Ennore in Madras 69 *A. culicifacies* gave 6 infected, while 364 *A. rossii* gave none. Bentley (1911) in Bombay dissected 837 *A. stephensi* and 772 *A. rossii*, and found 84 of the former had oöcysts and none of the latter. As regards sporozoites, of 826 *A. stephensi* 29 showed these, and of 680 *A. rossii* none. Darling (1909a) found in Panama that *A. albimanus*, *A. argyritarsis*, and *A. tarsimaculatus* could be infected with malaria, and that *A. malefactor* (*A. punctimacula*) could not.

As to whether any special mosquito is more susceptible to one particular type of malarial parasite there is little precise evidence. It was suggested in Macedonia that *A. maculipennis* was possibly the chief carrier of *P. vivax* and *A. superpictus* of *P. falciparum*. Experiments conducted by the writer (1921c) proved that both these mosquitoes readily became infected with both malarial parasites. In drawing conclusions from observations of this kind it has to be remembered that, as already noted, quinine has a greater influence on the gametocytes of *P. vivax* than on those of *P. falciparum* in preventing their subsequent development. Furthermore, in conducting comparative experiments, absolutely identical conditions must be employed, and this can only be done by feeding the mosquitoes to be compared at the same time, and keeping them afterwards under the same conditions. Kinoshita (1906), working in Formosa, stated that *P. falciparum*, which on this island depends on the presence of *A. listoni*, would not develop in *A. sinensis (A. hyrcanus)*, though *P. vivax* did so readily. The difficulty of obtaining accurate data is well illustrated by the experience of Mitzmain (1916). He fed 219 specimens of *A. punctipennis* on a *P. falciparum* case, and no infections occurred, whereas *A. quadrimaculatus* and *A. crucians*, fed at the same time, easily became infected. *A. punctipennis* was readily infected with *P. vivax*. In a later experiment, Mitzmain (1917) again fed *A. punctipennis* on a *P. falciparum* case, and obtained 27 per cent. of infections.

Swellengrebel, Schüffner and Swellengrebel de Graaf (1919) conducted experiments in the Dutch East Indies with a number of anopheles, with the object of testing their relative susceptibility to the three species of malarial parasite. The mosquitoes were fed on cases containing gametocytes on at least two occasions, and were examined for infection after ten to twelve days. The results obtained are shown in the following table, the first figure representing the number of mosquitoes examined after feeding, and the second the percentage found infected:

	A. ludlowi.		A. sinensis. (A. hyrcanus).		A. umbrosus.		A. barbirostris.		A. abolænidus.		A. indefinita (A. subpictus).		A. punctulatus.		A. leucosphyrus.		A. kochi.	
P. falciparum	37	100	292	5	87	0	10	0	6	0	7	0	23	4·3	3	0	5	0
P. vivax ..	47	80	175	4·06	104	5	15	13	1	0	5	0	10	0.9	0	6		16·7
P. malariæ ..	107	4·7	188	1	6	0	1	0	1	0	8	0	—	.2	0	—		—

It will be noted that in the case of *A. ludlowi*, 100 per cent. became infected with *P. falciparum*, 80 per cent. with *P. vivax*, and only 4·7 per

cent. with *P. malariæ.* In attempts to obtain data of this kind, it has always to be remembered that gametocytes in different cases may not all be in the same condition of readiness for development.

The number of anopheles found infected in nature depends on the intensity of infection amongst the population. Statements have been made that in certain localities as many as 50 per cent. or even 100 per cent. of the anopheles are infected. It must be very doubtful if these figures are correct. In Macedonia, in a very heavily infected village, the writer (1921c) found from dissections of 2,910 anopheles the following percentages infected: November, 1917, to February, 1918, 0·5; March, 1918, to June, 1918, 0·3; July, 1918, to October, 1918, 1·5; November, 1918, 0·2. These figures agree fairly well with those obtained by other observers. From the point of view of malaria prevention, the important point is not so much whether any particular anopheles can be experimentally infected, but to what extent it is infected under natural conditions. It may be accepted that a natural infection of anopheles as high as 10 per cent. is very exceptional.

Anopheles in which Oöcysts or Sporozoites have been Found either Naturally or Experimentally.

NOTE.—When more than one name for a mosquito is quoted, the first is that under which the record of infection was given by the observers mentioned. The names in brackets are synonyms, while those without brackets are the correct names according to Christophers (*Indian Medical Research Memoirs*, No. 3, December, 1924).

Anopheles sp. (" dappled-winged mosquito "): Ross, 1897 and 1898, India.

Anopheles sp.: Ziemann, 1900, West Africa, Cameroons.

A. aconitus: Swellengrebel and S. de Graaf, 1911, Dutch Indies; Stanton, 1914, Malay States; Barber, 1918, Malay States; Schüffner, 1918, Sumatra; Winoto, 1918, Java; Swellengrebel, 1919, Java; Walch and Walch-Sorgdrager, 1921, Sumatra; Doorenbos, 1925, Dutch East Indies.

A. albimanus: Darling, 1909, Panama; Laidislao and Chagas (quoted by Neiva, 1909), Brazil.

(*A. albipes*)=*A. albimanus:* Gray and Low, 1902, St. Lucia (very doubtful; dissection only forty-eight hours after feeding).

(*A. albirostris*)=*A. aconitus:* James and Stanton, 1912, Malay States; Stanton, 1912, Malay States.

A. algeriensis: Sergent, 1905, Algeria.

(*A. annulipes*)=*A. leucosphyrus:* Kinoshita, 1906, Formosa.

(*A. arabiensis*)=*A. gambiæ:* Patton, 1905, Aden Hinterland.

A. argyritarsis: de Faria and Laidislao (quoted by Neiva, 1909), Brazil; Darling, 1910, Panama; Boyd, 1925, Rio de Janeiro.

A. austeni: Wellman and Fay, 1907, Portuguese West Africa.

A. barbirostris: Stephens and Christophers, 1902, India; Walker and Barber, 1914, Philippines; Walker, 1915, Philippine Islands; Barber, 1918, Malay States; Schüffner, 1918, Sumatra; Swellengrebel, 1918, Dutch Indies; Swellengrebel, Schüffner, and S. de Graaf, 1919, Dutch Indies.

A. bellator, Davis, 1926, Brazil.

A. bifurcatus : Grassi, Bignami, and Bastianelli, 1899, Italy; Blacklock and Carter, 1920, England; Hargreaves (quoted by Robertson, 1920), Italy (Taranto); Yorke and Macfie, 1924, England.

A. brasiliensis : Godoy and Pinto, 1923, Brazil.

(*A. christophersi*)=*A. minimus :* Stephens and Christophers, 1902, India.

(*A. cohæsus* and *A. formosaensis*)=*A. minimus :* Tsuzuki, 1902, Formosa.

(*A. costalis*) ? *A. cinereus :* Hill and Hayden, 1905, Natal; Ross, 1908, Mauritius.

(*A. costalis*)=*A. gambiæ :* Ross, Annett, and Austen, 1900, West Africa; Stephens and Christophers, 1900, West Africa; Annett, Dutton, and Elliott, 1901, Nigeria (or *A. funestus*); Dutton, 1902, Gambia; Ziemann, 1902, Cameroons; Lamborn, 1925, Nyasaland.

A. crucians : Beyer, Pothier, Couret, and Lemann, 1902, North America; Mitzmain (Bruce Mayne), 1916 and 1919, North America; King, 1916, North America; Metz, 1919, North America.

A. culicifacies : Stephens and Christophers, 1902, India; James, 1902, India; Hodgson, 1912, India; James, 1913, Ceylon; Sinton, 1917, India; Gill, 1925, India.

(*A. febrifer*)=*A. minimus :* Walker and Barber, 1914, Philippine Islands.

(*A. fluviatilis*)=*A. listoni :* James, 1902, India.

(*A. formosaensis* and *A. cohæsus*)=*A. minimus :* Tsuzuki, 1902, Formosa.

A. fuliginosus : Stephens and Christophers, 1902, India; James, 1902, India; Adie, 1903, India; Lalor, 1911, India; Christophers, 1911, India; Hodgson, 1912, India; Stanton, 1912, Malay States; Stuart and Proctor (quoted by Fry, 1914), India; Fry, 1914, India; Bentley (quoted by Fry, 1914), India; Barbor, 1918, Malay States; Schüffner, 1918, Sumatra; Swellengrebel, Schüffner, and S. de Graaf, 1919, Dutch Indies.

(*A. funesta* var. *listoni*)=*A. listoni :* Perry 1912, India.

A. funestus: Ross, Annett, and Austen, 1900, West Africa; Stephens and Christophers, 1900, West Africa; Daniels, 1901, British Central Africa; Annett, Dutton, and Elliott, 1901, Nigeria (or *A. costalis*); Ziemann, 1902, West Africa, Cameroons; Dutton and Todd, 1906, Congo; Wellman and Fay, 1907, Portuguese West Africa; Newstead, Dutton, and Todd, 1907, Congo; MacGregor, 1924, Mauritius; Lamborn, 1925, Nyasaland.

A. hispaniola : Sergent, Ed. and Et., 1905, Algeria.

(*A. hunteri*)=*A. separatus :* Barber, 1918, Malay States.

(*A. indefinita*)=*A. subpictus :* Swellengrebel, 1918, Java; Schüffner, 1918, Sumatra; Swellengrebel, Schüffner, and S. de Graaf, 1919, Sumatra; Barber, 1918, Malay States;.

A. intermedius : Neiva and Laidislao (quoted by Neiva, 1909), Brazil.

(*A. jamesii*)=*A. maculipalpis :* Stephens and Christophers, 1902, India.

(*A. jesoensis*)=*A. hyrcanus :* Tsuzuki, 1902, Japan.

A. karwari : Barber, 1918, Malay States.

A. kochi : Barber, 1918, Malay States; Schüffner, 1918, Sumatra; Swellengrebel, 1918, Dutch Indies; Swellengrebel, Schüffner, and S. de Graaf, 1919, Dutch Indies; Walch and Walch-Sorgdrager, 1921, Sumatra. Doorenbos, 1925, Dutch East Indies.

A. leucosphyrus: Bais, 1919 (quoted by Swellengrebel and S. de Graaf, 1920), Sumatra.

A. listoni, Stephens and Christophers, 1901, India; Kinoshita, 1906, Formosa; Horne, 1914, India; Chalam, 1923, Assam.

A. ludlowi : Banks, 1907, Philippines; Christophers, 1912, Andaman Isles; Horne (quoted by Hodgson, 1914), Madras; Barber, 1918, Malay States; Swellengrebel, 1918, Dutch Indies; Swellengrebel, Schüffner, and S. de Graaf, 1919, Dutch Indies; Van Breemen, 1919, Batavia; Winoto, 1919, Java; Darling, 1920, Java; Walch and Walch-Sorgdrager, 1921, Sumatra; Schüffner and Hylkema, 1922, Sumatra; Rodenwaldt and Essed, 1925, Batavia.

A. lutzi : Galli-Valerio, 1904 (very doubtful), Brazil. (Only a few alcohol specimens examined.)

A. maculatus: Watson, 1911, Malay States (called *A. willmori*): Stanton, 1912, Malay States; Walker and Barber, 1914, Philippines; Walker, 1915, Philippines; Barber, 1918, Malay States; Essed, 1925, and Doorenbos, 1925, Dutch East Indies.

A. maculipalpis: Robertson, 1911, India; MacGregor, 1924, Mauritius.

A. maculipennis: Grassi, Bignami, and Bastianelli, 1898, Italy; van der Scheer and van Berlekom, 1901, Holland; Schaudinn, 1902, Italy; Schoo, 1902, Holland; Martirano, 1902, Italy; Jancsó, 1904, Germany; Sergent, 1905, Algeria; Cardamatis, 1906, Greece; Mollow, 1910, Bulgaria; Macdonald, 1911, Spain; Léger, 1913, Corsica; James, 1917, England; Cot and Hovasse, 1917, Macedonia; Roubaud, 1917, France; Blanc and Heckenroth, 1918, Albania; Joyeux, 1918, Macedonia; Grassi and Sella, 1920, Italy; Hargreaves (quoted by Robertson, 1920), Italy (Taranto); Wenyon, 1921, Macedonia; Roubaud and Léger, 1921, Corsica; Sergent, Parrot, and Donatien, 1921, Corsica; Swellengrebel, 1921, Holland; Yorke and Macfie, 1924, England; Mühlens and Sfarcic, 1925, Dalmatia.

(*A. nursei*)=*A. superpictus*: Mackie (quoted by Christophers and Shortt, 1921), Persia.

(*A. paludis*)=*A. mauritianus*: Christophers, 1900, Sierra Leone.

A. pharoensis: Dutton and Todd, 1916, Congo (only two dissected) ?; Manson-Bahr, 1918, Egypt.

A. plumbeus: Blacklock and Carter, 1920, England.

A. pseudomaculipes: Neiva and Laideslao (quoted by Neiva, 1909), Brazil.

(*A. pseudopictus*)=*A. hyrcanus*: Grassi, 1899, Italy.

A. pseudopunctipennis: Darling, 1909, Panama.

A. pulcherrimus: Christophers, 1921, Mesopotamia (Busra).

A. punctipennis: Dupree, 1905, North America; King, 1916, North America; Mitzmain, 1917, North America.

A. punctulatus: Swellengrebel, 1918, Java; Schüffner, 1918, Sumatra; Swellengrebel, Schüffner, and S. de Graaf, 1919, Dutch Indies; Walch and Walch-Sorgdrager, 1921, Sumatra; Heydon, 1923, New Britain; de Rook, 1924, New Guinea.

A. quadrimaculatus: Beyer, Pothier, Couret, and Lemann, 1902, North America; Hirschberg, 1904, North America; Mitzmain, 1916, North America; King, 1916, North America; Metz, 1919, North America.

(*A. rossii*)=*A. subpictus*: Stephens and Christophers, 1902, India; James, 1902, India; Schüffner, 1902, Sumatra; Vögel, 1909, Dutch Indies; Walker and Barber, 1914, Philippines; Barber, 1918, Malay States; Swellengrebel, 1919, Dutch Indies; Swellengrebel, Schüffner, and S. de Graaf, 1919, Dutch Indies; Van Breemen, 1919, Batavia; Reylingh (quoted by Swellengrebel and S. de Graaf, 1920), Java; Gill, 1925, India; Rodenwaldt and Essed, 1925, Batavia.

(*A. rosii* var. *indefinita*)=*A. subpictus*: Walker, 1915, Philippine Islands.

(*A. sinensis*)=*A. hyrcanus*: Stanton, 1912, Malay States; Kinoshita, 1906, Formosa; Barber, 1918, Malay States; Schüffner, 1918, Sumatra; Swellengrebel, 1918, Dutch Indies; Swellengrebel, Schüffner, and S. de Graaf, 1919, Dutch Indies; Walch and Walch-Sorgdrager, 1921, Sumatra; de Rook, 1923, Sumatra; Walch, 1924, Sumatra; Doorenbos, 1925, Dutch East Indies.

A. stephensi: Stephens and Christophers, 1902, India; James, 1902, India; Liston, 1908, India; Bentley, 1911, India; Hodgson, 1912, India; Sinton, 1917, India; Gill, 1925, India.

A. superpictus: Grassi, Bignami, and Bastianelli, 1899, Italy; Cardamatis, 1906, Greece; Cot and Hovasse, 1917, Macedonia; Wenyon, 1921, Macedonia; Christophers, 1921, Mesopotamia (Musul).

A. tarsimaculatus: Darling, 1909, Panama.

A. tesselatus: Swellengrebel and Schüffner, 1919, Java.

A. theobaldi: Stephens and Christophers, 1902, India.

A. turkhudi: Stephens and Christophers, 1902, India; James, 1902, India; Manson-Bahr, 1918, Egypt. (Manson-Bahr's record really refers to *A. multicolor*.)

A. umbrosus : Roper, 1914, North Borneo; Barber, 1918, Malay States; Swellengrebel, 1918, Dutch Indies; Swellengrebel, Schüffner, and S. de Graaf, 1919, Dutch Indies.

A. vagus : Doorenbos, 1925, Dutch East Indies.

A. willmori : Watson, 1911, Malay States (really *A. maculatus*); Adie, 1911, India; Gill, 1923, Murree, Himalayas.

Cultivation of Malarial Parasites.

Bass and Johns (1912) were the first to succeed in cultivating malarial parasites. This was most easily accomplished in the case of *P. falciparum*, which was observed to pass through three generations of schizogony in these cultures. The procedure is to withdraw 10 c.c. of malarial blood from a vein, add to it 0·1 c.c. of a 50 per cent. solution of dextrose, and defibrinate the mixture. The blood is distributed in test-tubes, which are then incubated at 40° C.; the cells settle to the bottom, leaving a column of serum about 2·5 cm. in height. The parasites will be found in the red cells just below the surface of the deposit, and by examining them at intervals it will be seen that they grow into mature schizonts and break up into merozoites. Some of these may enter other cells, and again grow into schizonts, but as a rule, so soon as the merozoites escape from the cells, they are phagocyted by the leucocytes. If the original mixture, after defibrination, is centrifuged, the leucocytes can be removed, as they form the upper layer of the deposit. In this way, unhampered by leucocytes, the parasites may complete two or three cycles. For subcultures, normal blood is taken and treated in the same manner, after which it is inoculated by means of a pipette with infected blood from the previous culture. In all these processes it is absolutely essential to avoid bacterial contaminations. Ziemann (1913, 1914) modified the original method in details, and claimed that better results were obtained by adding double the quantity (0·2 c.c.) of the dextrose solution. Culture of malarial parasites has also been successfully carried out by Thomson, J. G. and D. (1913), Rocha-Lima and Werner (1913), McLay (1922), and others.

If cultures are commenced with blood containing ring forms, as is generally done, these grow into the schizonts, which produce merozoites. The rate of growth varies with the temperature. At 41° C. *P. vivax* takes about forty-eight hours, whereas *P. falciparum* occupies from twenty-four to forty-eight hours. The development as regards the action on red blood-corpuscles, production of Schüffner's and Maurer's dots, and the number of merozoites corresponds with what occurs in the human body. Beside the normally reproducing parasites, there occur others, evidently abnormal or degenerate forms.

In the case of *P. falciparum*, Thomson, J. G. and D., noted that there was a tendency for the infected cells to clump together in the culture, a fact which may have some bearing on the formation of emboli of infected cells in the capillaries of the brain and other organs in severe cases of malignant tertian malaria. McLay (1922) also noted that the infected red cells tended to collect around the large mononuclear cells (Fig. 401). As these are probably of endothelial origin, this observation may also explain the tendency of infected cells to accumulate as emboli in the capillaries. In sections of heavily infected organs the smallest capillaries are completely blocked, whereas the vessels with a slightly larger diameter are clear at the centre, but show accumulations of parasites against the endothelial cells peripherally. McLay also noted that it was difficult, if not impossible, to obtain cultures from cases which were taking quinine.

In culture some have observed what they consider to be the formation of gametocytes, while others, as, for instance, Joukoff (1913) and Perekropoff (1914), claim to have seen the mosquito cycle. Perekropoff, by using 0·4 c.c. of dextrose solution instead of the usual 0·1 or 0·2 c.c., claims to have followed the sporogony cycle of the gametocytes of *P. falciparum*. He gives figures of what he considers oöcyst formation within the red blood-corpuscles, with production of sporozoites, but there is no real evidence that he was dealing with any other than a schizogony cycle modified by the artificial conditions of culture.

When Bass first announced the successful culture of malarial parasites, it was hoped that a method had been discovered of maintaining them indefinitely. This hope, however, has not been realized, for the cultures are difficult to maintain, as growth ceases after a very few subcultures. Nothing comparable with the successful culture of trypanosomes and leishmania has been accomplished.

PLASMODIA OF MONKEYS.

A malarial parasite of monkeys was first noted by Koch (1898a). It was described by Kossel in 1899, and named *Hæmamœba kochi* by Laveran (1899), who saw it in *Cercopithecus sabœus*. It was discovered by Koch in Africa in various species of *Cynocephalus*, *Cercopithecus*, and *Cercocebus*, and Lühe (1906) refers to this species a form seen by Ziemann in the chimpanzee. It was next seen by Bruce and Nabarro (1903), and by Dutton, Todd and Tobey (1906) in Africa in species of *Cercopithecus*. Ross, P. H. (1907), saw it in monkeys in Uganda. Gonder and Berenberg-Gossler (1908) studied it in *Cercopithecus fuliginosus* which had been imported to Hamburg from Africa. Sergent (1908) saw it again in *Cercopithecus albogularis*; Martoglio, Stella and Carpano (1910) in *Cercopithecus sabœus*;

Ringenbach (1914) in *Cercopithecus cephus ;* and Plimmer (1916) in *Cerco-cebus œthiopicus.* Seidelin and Connal (1914) recorded the presence of what may be this form in *Cercopithecus mona* and *Papio sphinx* of West Africa. Halberstädter and Prowazek (1907) described two species from Borneo, one *Plasmodium pitheci* of the orang-outang, first seen by Laveran (1905*b*) in an imported animal in Paris, and the other *P. inui* of *Macacus cyno-molgus* and *M. nemestrinus*, which is probably the form seen by Bruce and Nabarro (1903) in *M. rhesus.* *P. pitheci* was inoculable to other orang-outangs, but not to the lower monkeys, while *P. inui* was inoculable to species of *Macacus*, but not to orang-outangs. The form seen by Chimisso (1922) in a *M. rhesus* in Italy is probably *P. inui.* Mayer (1908) described as *P. cynomolgi* a parasite he found in *M. cynomolgus.* It was inoculable to *M. cynomolgus*, *M. rhesus*, and a *Cercopithecus*, and is probably identical with *P. inui.* What was probably the same organism was seen by Mathis and Léger (1911) in *M. rhesus* and *M. lasiotis tcheliensis* in Tonkin. They found it was easily inoculable to monkeys of these species. Blanchard and Langeron (1913), who found an imported monkey (*M. cynomolgus*) naturally infected in Paris, succeeded in infecting two other monkeys of the same species. One died of an acute infection, the other acquiring a chronic one. This strain was further studied by Bouniol, whose results were published after his death by Blanchard and Langeron (1913*a*). He noted that the infections were either acute ones which proved rapidly fatal, or chronic ones of many months' duration, the monkeys ultimately dying of some intercurrent malady. He showed that the infected red cells developed Schüffner's dots, and that the cycle of development occupied forty-eight hours. Léger and Bouilliez (1912) inoculated *P. inui* to *M. cynomolgus*, *M. sinicus*, *M. rhesus*. *M. nemestrinus*, *Cercopithecus callitrichus*, *C. patas*, *C. cebus*, and *Papio anabis.* One *C. fuliginosus* and two chimpanzees were refractory. Donovan (1920) saw this form in *M. sinicus*, and refers it to *P. cynomolgi.* A form (*P. brasilianum*) was described by Gonder and Berenberg-Gossler (1908) from *Brachyurus calvus* in South America, while Knowles (1919) gave the name of *P. semno-pitheci* to a form seen by him in the Indian hanuman monkey (*Semno-pithecus entellus*). Finally, Reichenow (1917*a*) described malarial parasites in anthropoid apes. He regarded these as identical with the human parasites, but Sluiter, Swellengrebel and Ihle (1922) proposed the name *Laverania reichenowi* for the form in the chimpanzee.

Plasmodium reichenowi (Sluiter, Swellengrebel and Ihle, 1922).— Reichenow (1917*a*) published a preliminary account of observations on plasmodia of anthropoid apes studied by him in the Cameroons (Plate XV., 1-7, p. 970). He regarded them as identical with the three human species. In the same year Mesnil and Roubaud announced the successful inoculation

of *P. vivax* to the chimpanzee, a result which was of considerable interest in the light of Reichenow's conclusions, and the fact that previous attempts by Koch and others to inoculate human malaria to these animals had been unsuccessful. Reichenow (1920c) has published a fuller account of the malarial parasites seen by him in the chimpanzee and gorilla. He has shown that these apes live in the vicinity of human habitations, and are liable to be bitten by infected mosquitoes. In adult apes the parasites are scanty, and can only be demonstrated by the thick film method. In young animals they are more numerous. Forms corresponding completely with *P. falciparum* were seen. In these the rings were small, the schizonts did not completely fill the red cell, and the gametocytes were of the typical crescent form. They resembled in every way the *P. falciparum* found in the negro. Another type resembled *P. vivax*. The rings were large, there was enlargement of the red cell, with production of typical schizonts and gametocytes, while the cycle of development occupied forty-eight hours. Rings, schizonts, and gametocytes of the *P. malariæ* type were also seen, as well as band forms, which are fairly characteristic of this species. It would seem, therefore, that Reichenow has produced definite evidence in support of his contention that the human malarial parasites of man occur in the anthropoid apes of Africa. The successful inoculation of *P. vivax* to the chimpanzee by Mesnil and Roubaud is a further argument in favour of his conclusions. Reichenow believes that the form seen by Ziemann in the chimpanzee and referred to *P. kochi* by Lühe (1906) may in reality be one of the human forms, as also *P. pitheci* of the orang-outang of Borneo.

The parasites of the chimpanzee have also been studied by Blacklock and Adler (1922) in West Africa. They observed in the blood of the animal large amoeboid forms like those of *P. vivax* in enlarged and pale red cells, large more or less band forms with coarse pigment like *P. malariæ*, small ring forms in cells of normal size and colour resembling ring forms of *P. falciparum*, and gametocytes of crescent form indistinguishable from those of *P. falciparum*. No segmenting forms were seen in the blood. Attempts to infect two human beings by inoculations of blood both subcutaneously and intravenously failed, nor did the mosquito, *Anopheles costalis*, become infected after feeding on the chimpanzee. In a later paper, Blacklock and Adler (1924) state that they failed to infect a young chimpanzee by inoculating human blood containing *P. falciparum*. A further examination of thirteen chimpanzees in Sierra Leone by Adler (1923) revealed an infection only in two young animals. Gametocytes and young ring forms, like those of *P. falciparum*, were the only stages seen. The livers of both these apes showed a high degree of fatty degeneration. As noted above, the parasite which resembles *P. falciparum* was named *Laverania reichenowi*.

PLATE XV.

PLATE XV.

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B.F.

Plasmodium kochi (Laveran, 1899).—This species is a common parasite of monkeys belonging to the genera *Cercopithecus, Cynocephalus*, and *Cercocebus* in tropical Africa. It has been described from the chimpanzee, but, as already remarked, Reichenow thinks that the form in this animal is another species (Plate XV., 22-28, p. 970).

The asexual cycle of *P. kochi* is completed in forty-eight hours. In this and in other respects it resembles *P. vivax*. The ring forms are large, but some of them occupy a smaller proportion of the red cell than rings of *P. vivax*. As growth proceeds, the resemblance to *P. vivax* becomes more marked. The red cell enlarges, while the parasite becomes irregular in shape, though not to the same extent as the human parasite. The pigment granules are of a light brown colour, while sometimes a stippling of the cell like Schüffner's dots occurs. The fully-formed schizonts produce from eight to fourteen merozoites, and bear a striking resemblance to those of *P. vivax*. The gametocytes are large round bodies, which may be distinguished as male and female gametocytes, as in the human parasite. Microgamete formation by flagellation has been observed, but no development could be obtained by Gonder and Berenberg-Gossler (1908) in *Anopheles maculipennis*. The parasite shows little sign of pathogenicity in natural infections or in inoculated animals. Gonder and Rodenwaldt (1910) noted, however, that if splenectomy had previously been performed, the infections were much more severe, elevations of temperature occurred, and the parasites persisted in the blood for many months. Two unsuccessful attempts were made to inoculate the parasite into human beings. It was observed that quinine had a definite action in controlling the infections. Leger, M. (1922), has described as *P. bouilliezi* a parasite found in the blood of a freshly killed *Cercopithecus campbelli* of West Africa. It is said to differ from *P. kochi* in that the young forms are elongate and not annular, while the schizonts, or at least the forms described as such, are smaller. It is evident that not sufficient material was studied to justify the separation of the parasite from *P. kochi*.

Plasmodium inui Halberstädter and Prowazek, 1907.—This parasite was discovered by Halberstädter and Prowazek in monkeys (*Macacus*) in Borneo. The form studied by Mayer, and named by him *P. cynomolgi*, is probably identical with *P. inui*. It resembles *P. vivax* in most of its stages, producing enlargement of the red cell, Schüffner's dots, and a yellow-brown pigment (Plate XV., 8-14, p. 970). Some of the ring forms, however, are small and resemble those of *P. falciparum*, while the gametocytes are not much larger than the normal red cell and resemble those of *P. malariæ*. Mayer (1908) could obtain no development in *Culex pipiens* nor in *Aëdes argenteus*. He believes that he observed small oöcysts in *Anopheles maculipennis*, but the complete development was not followed.

As already remarked, Leger and Bouilliez (1913) inoculated *P. inui* to a number of monkeys, including members of the genus *Cercopithecus*, the common hosts of *P. kochi*. Many of the monkeys died of heavy infections. Quinine, even in large doses, appeared to have no influence on the infection.

Plasmodium semnopitheci Knowles, 1919.—Knowles (1919) records that a monkey (*Semnopithecus entellus*) which had been under laboratory observation for a considerable time was inoculated intravenously with 2 c.c. of human blood containing crescents of *P. falciparum*. The monkey became ill twenty-four hours later, and died forty-eight hours after the injection. It had a very intense malarial infection, and as any young forms inoculated could not in so short a time have given rise to those seen, it was rightly concluded that the injection had stimulated to sudden development a latent infection which the monkey already possessed. Nearly every red cell was found infected, and many of the parasites appeared to be extracellular and free in the plasma. Small rings of the *P. falciparum* type were found, as also larger rings like those of *P. vivax*. There was definite enlargement of the red cells, but the schizogony stages were not seen. The gametocytes were large and of the *P. vivax* type. Though the parasite has been given a distinct name, it seems to be very similar to *P. inui*. The intensity of the infection and the fact that the examination was made after death would account for the occurrence of extracellular and other abnormal forms.

Plasmodium pitheci Halberstädter and Prowazek, 1907.—This parasite of the orang-outang (*Simia satyrus*) of Borneo, resembles *P. inui* very closely, but is distinguished from it by its pigment, which is dark brown or black in colour (Plate XV., 29-35, p. 970). It is inoculable to other orang-outangs, but not to the lower monkeys (*Macacus*), which are very susceptible to *P. inui*. Shibayama (1910) states that Schüffner's dots are not present, but from his figures it would seem that the staining was not sufficiently intense to make them evident. Reichenow (1920c) considers it possible that *P. pitheci* may be one of the human parasites. Dodd (1913) records an infection of an orang-outang with *P. pitheci* which proved fatal.

Plasmodium brasilianum Gonder and Gossler, 1908.—This is a parasite discovered by Gonder and Berenberg-Gossler (1908) in a monkey (*Brachyurus calvus*) which had been imported to Hamburg from the Amazon district. In contrast to the forms described above, which resemble *P. vivax*, this one shows a greater similarity to *P. malariæ* (Plate XV., 15-21, p. 970). There is no enlargement of the red cells, and the schizonts, which give rise to six or twelve merozoites, completely fill the cell. Band forms occur, as in *P. malariæ*, and the gametocytes are also of the type characteristic of this species.

Seidelin (1912) described a parasite, probably a plasmodium, which he had found in the blood of a monkey, *Ateles* sp. of Yucatan. Only small unpigmented rings resembling those of *P. falciparum* were seen.

PLASMODIA OF BATS AND OTHER SMALL MAMMALS.

Plasmodium murinum (Dionisi, 1899) and **P. melanipherum** (Dionisi, 1899).—The occurrence of pigmented intracorpuscular parasites in bats was first made known by Dionisi (1898). He later (1899) described two species of pigment-producing plasmodia under the names of *Polychromophilus murinus* and *P. melanipherus*. Another form which was devoid of pigment was named *Achromaticus vesperuginis*. The pigment-producing forms undoubtedly belong to the genus *Plasmodium*.

P. murinum has been described from *Vespertilio murinus* in Italy by Dionisi (1899), from *V. capensis* of South Africa by Bowhill (1906), and from *V. daubentoni* of Russia by Schingareff (1906). *P. melanipherum* was first noted in *Miniopterus schreibersi* in Italy by Dionisi (1898), by Sambon and Low (1901) in *Myotis capaccinii* in Italy, then by Schingareff (1906) in the same host in Russia. What was presumed to be a variety of the same organism was described under the name *P. melanipherum* var. *monosoma* by Vassal (1907) from *Vesperugo abramus* of Annam. Dutton, Todd, and Tobey (1906) record a similar form from undetermined bats of the Congo. Generally speaking, the above-named species resemble *P. malariæ*, and Dionisi separated the two species on account of differences in arrangement of the chromatin granules (Plate XVI., 23-27, p. 974). Forms which are undoubtedly gametocytes occur, and some observers have noted rounded bodies with as many as ten chromatin dots, giving the appearance of schizonts of the *P. malariæ* type. Many curious forms have been described by Dionisi, and they were named by him *Polychromophilus* on account of the red staining of many of the granules. It is evident that all these forms require further investigation in the light of present knowledge.

The non-pigmented parasite first described by Dionisi as *Achromaticus vesperuginis* is probably a piroplasma (Plate XVI., 28-32, p. 974).

Plasmodium brodeni Rodhain, Pons, Vandenbranden, and Bequaert, 1913.—This parasite was discovered by Rodhain and his co-workers in the Belgian Congo in the jumping rat, *Petrodromus tetradactylus*. The forms seen are of the *P. malariæ* type, except that a slight enlargement of the red cell may occur. Only ring forms, young schizonts, and gametocytes were seen.

A very similar form (*P.* sp.) was described by Rodhain (1915) from the same region in the flying fox, *Epomorphus franqueti*, while A. and M. Leger

(1914) saw what was probably the same parasite in *E. gambianus* in Upper Senegal. The latter observers identified the parasite with *P. pteropi*.

Plasmodium pteropi Breinl, 1912.—This is a parasite of the *P. malariæ* type which was discovered by Breinl in West Australia in the blood of the flying fox, *Pteropus gouldi*. Only ring forms, immature schizonts, and gametocytes were seen. The pigment, judging from the coloured plate accompanying the description, is of a light brown colour, and resembles that of *P. vivax* rather than that of *P. malariæ*. Some slight enlargement of the infected cells may occur.

Mackie (1914a) in India described a very similar form from the flying fox, *Pteropus edwardsii*. He gave it the name *Plasmodium pteropi* without knowing that the name had been employed by Breinl for the West Australian form. It is very probable, however, that the two are identical. What is probably the same parasite was seen by Manson-Bahr in the flying fox in Ceylon (Plate XVI., 8-12, p. 974).

Plasmodium vassali (Laveran, 1905).—This parasite was discovered by Vassal (1905a) in the squirrel, *Sciurus grisemanus*, of Annam, and was named *Hæmamœba vassali* by Laveran (1905b). Later, Vassal (1907a) found it also in two other species, *S. vittatus* and *S.* sp. In many respects the organism resembles *P. malariæ*, but no actual mature schizogony forms were seen, the only ones occurring being gametocytes, ring forms, partially grown gametocytes, and possibly young schizonts (Plate XVI., 18-22, p. 974). A method of reproduction of the smaller forms by binary fission is described. The male forms flagellate very readily, but no development was obtained in mosquitoes (*Anopheles, Culex*), or other flies (*Tabanus, Hæmatopota*, or *Hippobosca*). The parasite was not inoculable to man, monkeys, rabbits, guinea-pigs, rats, mice, or the European squirrel, *Sciurus vulgaris*.

A similar form was seen by Donovan (1920) in India in the Malabar squirrel, *Ratufa indica*. It was named by him *Plasmodium ratufæ*.

The Royal Society's Commission, under Bruce (1915a), record the occurrence of a plasmodium in the jumping shrew of Nyasaland. Only gametocytes containing a single compact nucleus and scattered pigment were seen. They were definitely larger than the normal red cells.

Plasmodium roubaudi Leger and Bédier, 1923.—This parasite was discovered in one out of five zorillas (*Ictonyx zorilla*, one of the Mustelidæ) examined in Senegal by Leger and Bédier (1923). The smallest forms are round bodies about 1·25 microns in diameter. They contain one or two dots of chromatin and a small vacuole, which gives them a more solid appearance than the ring forms of the well-known parasites of malaria. The pigment granules are not numerous. Narrow rod-like forms 3·5 microns in length by 0·4 to 0·5 microns in breadth, and larger ovoid

PLATE XVI.

Malarial Parasites of Various Mammals as seen in Dried Blood-Films stained with Romanowsky Stains. (× 2,000).

1-7. *Plasmodium cephalophi* of the duiker.
8-12. *Plasmodium pteropi* (?) from the flying fox of Ceylon.
13-17. *Plasmodium bubalis* of the Indian ox.
18-22. *Plasmodium vassali* in the squirrel of Annam.
23-27. *Plasmodium murinum* from the bat.
28-32. *Babesia vesperuginis* of the bat.

1-7. after Bruce, Harvey, Hamerton, and Lady Bruce; 8-12. from Films presented by Dr. Manson-Bahr; 13-17, from Original Drawings by Sheather; 18-22, from Films made from Infected Animals at the Pasteur Institute; 23-27. after Dionisi; 28-32. after Yakimoff, Stolnikoff, and Kohl-Yakimoff.)

PLATE LXVI.

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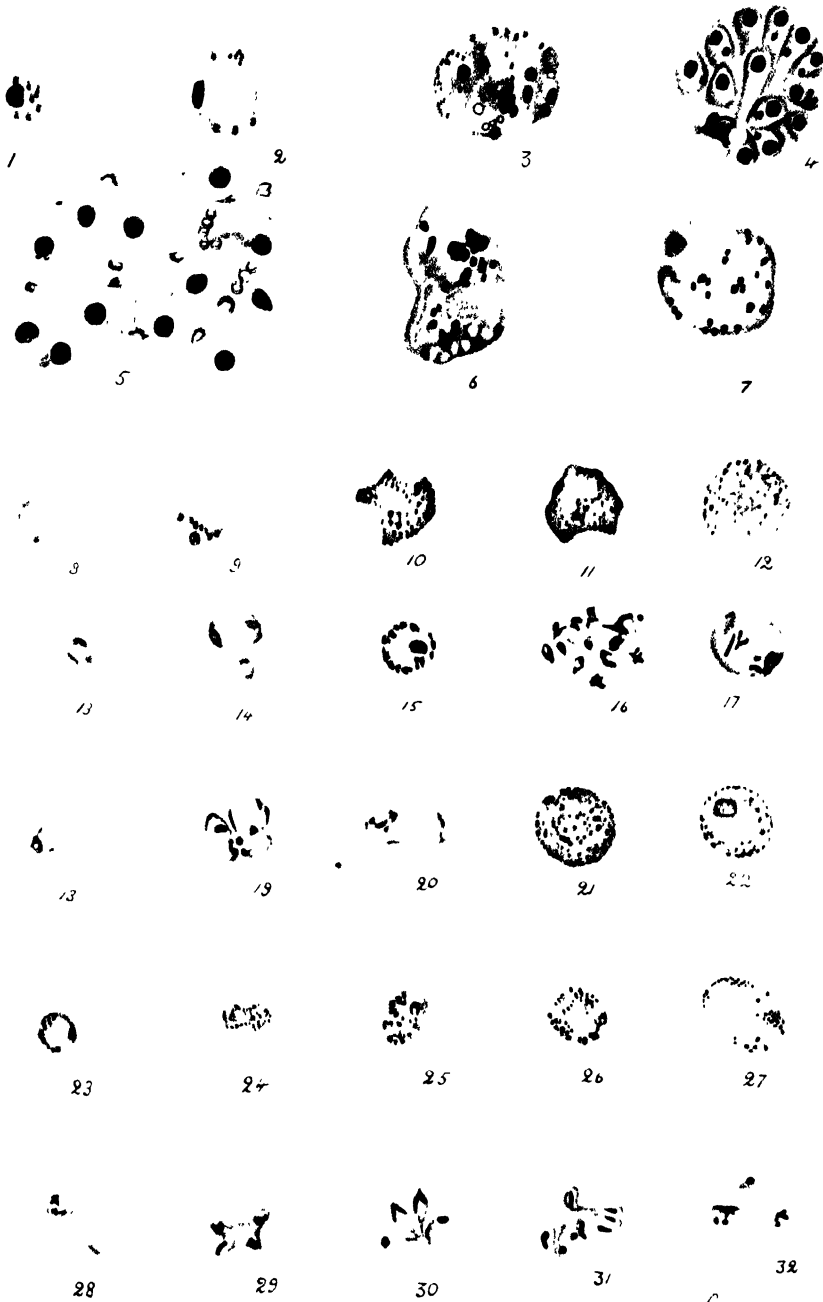
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forms which stretch across the cell as do the band forms of *P. malariæ*, also occur. These larger parasites contain definite pigment granules. No schizogony stages were seen. Another species recorded by Leger, M., and Bédier (1922*b*) is *P. rigoleti* of *Myoxus murinus,* also of Senegal. In this case the parasite occurred as small rings or amœboid bodies, but was devoid of pigment.

Thomson, J. D. (1906), described a new type of intracorpuscular parasite of the British mole. He stated that it occasionally contained granules of a black pigment. França (1911*a*) studied the organism in Portugal, and concluded that it was allied to the piroplasmata, and gave it the name *Elleipsisoma thomsoni* (see p. 1050).

PLASMODIA OF LARGE MAMMALS.

Plasmodium cephalophi Bruce, Harvey, Hamerton, and Lady Bruce, 1913.—This parasite was discovered by the Royal Society's Commission (Bruce *et al.,* 1913*c*) in the blood of two young duikers (*Cephalophus grimmi*) in Nyasaland (Plate XVI., 1-7, p. 974). In one animal an acute infection lasting only four days occurred, while in the other a less intense infection persisted for some months. In its compact form and number of merozoites (eight to twelve) the parasite showed resemblances to *P. malariæ* of man. There was, however, a marked enlargement of the cell with growth of the parasite. Schüffner's dots were not present, though in some cases a coarse stippling like Maurer's dots was seen. The red cells became very pale in colour. Another peculiarity is that the pigment, when it appears, is not scattered through the cytoplasm, but is collected in a single vacuole, giving the appearance of a brownish-yellow area in the parasite. Some of the large forms, which are evidently game-tocytes, had, in addition to the single nucleus, other red or purple staining granules in the cytoplasm.

In two other duikers another plasmodium was seen, which seemed to differ from *P. cephalophi* in that the pigment was aggregated into a denser clump. Whether it was actually a distinct species or a slightly modified form of *P. cephalophi* was not determined.

Plasmodium bubalis Sheather, 1919.—This form was discovered by Sheather in India in a buffalo which died after inoculations made for immunization purposes. There was a fairly heavy infection, 1·6 per cent. of the red cells containing parasites. *P. bubalis* resembles in many respects the human quartan parasite, *P. malariæ* (Plate XVI., 13-17, p. 974). Some of the ring forms are as small as those of *P. falciparum,* while a slight enlargement of the red cell is caused by some of the larger parasites which, when partially grown, have a regular outline. The adult schizont,

which appears to produce from seven to fourteen merozoites, completely fills the red cell, and has its pigment aggregated in a single clump. Some of the large forms with a single nucleus are probably gametocytes, though Sheather does not mention them. Many of these had a vacuole in the cytoplasm. Edwards (1925) has also seen the parasite in India. Kolle (1898) described as *P. bovis* a supposed malarial parasite of South African cattle. It is not clear that he was actually dealing with a plasmodium.

Plasmodium capræ (de Mello and Paes, 1923).—De Mello and Paes (1923) have described a pigmented parasite of the red blood-corpuscles of goats in Angola under the name *Laverania capræ*, on account of the fact that the gametocytes were crescent-shaped and resemble those of *P. falciparum*. The smallest forms were also like the ring stages of this parasite. They can be traced through a series of larger forms into schizonts, which give rise to six or seven or even thirteen merozoites. The gametocytes, which are crescent-shaped, can be distinguished as male forms measuring 5 to 6 by 2 to 2·5 microns, and female forms 3·5 to 4·5 by 1 to 2 microns. The red cells are little altered by the young stages of the parasite, but are distinctly paler than normal at the adult schizont stage. There was no development of Schüffner's or other dots. Paes (1924) again refers to the parasite, which he illustrates in a coloured plate. None of the forms depicted shows any pigment, and it is stated that this substance is not regularly present. The so-called gametocytes are actually pear-shaped extracellular bodies which are devoid of pigment, while the two figures illustrating schizogony are far from convincing. It seems possible that the organism is actually a species of *Babesia*.

Plasmodium canis Castellani and Chalmers, 1910.—This is a parasite which Castellani and Chalmers (1910) claim to have discovered in dogs in Colombo. The figures and description show that it is morphologically identical with *P. vivax* of man, so that it could easily be mistaken for this parasite if the source of the blood were not known. Castellani (1924) again refers to it, and states that he saw several cases of the infection in dogs in Colombo. Though it is said to be a very common parasite, no other observer has seen the organism. Owing to the kindness of Mr. Burgess of the Bacteriological Institute of Colombo, the writer obtained blood-films from 500 pariah dogs. In none of these could *P. canis* be discovered.

Plasmodium equi Castellani and Chalmers, 1913.—This form, like the above, is said to occur in Colombo. It was found in a horse, and is very similar to *P. canis*. It has been recorded by no other observer.

PLASMODIA OF BIRDS.

The malarial parasites of birds (Plate VI., 1-10, p. 882) are of special interest in connection with the investigations of Ross, R. (1898b), who demonstrated their complete development in mosquitoes and proved that birds become infected by the insects injecting sporozoites which collect in the salivary glands. It was these brilliant researches which revealed an entirely new cycle of development for Protozoa, and led directly to the discovery of an identical cycle for the parasites of human malaria.

Pigmented parasites in the blood-corpuscles of birds were first seen by Danilewsky, but Grassi and Feletti (1890) were the first to recognize that there occurred in birds a true malarial parasite, which they called *Hæmamœba præcox*, to distinguish it from halteridium (*Hæmoproteus*). Later in the same year Danilewsky (1890) recognized an acute disease in birds, but failed to differentiate the parasite from *Hæmoproteus*. Celli and Sanfelice (1891) gave a more accurate description of the organism, and were the first to inoculate it to other birds. Laveran (1891) also described it, while Danilewsky (1891) gave a detailed description of the disease produced by the parasite, which he called *Cytosporon malariæ avium*. Grassi and Feletti (1892) worked out the details of its development in birds and grouped it in their genus *Hæmamœba* with the similar parasite of man. Labbé (1894) gave it the name *Proteosoma*, which is still frequently used in a popular sense. Grassi and Feletti (1890) had employed the name *Hæmamœba præcox*, and this seems to be the first name given according to the rules of nomenclature. The same observers (1891) used the name *H. relicta*, which is often employed at the present time. As the parasite undoubtedly belongs to the same genus as those causing human malaria, the correct name for proteosoma, the parasite of bird malaria, is *Plasmodium præcox*. Some observers (Doflein, 1916, França, 1917) retain the generic name *Proteosoma* for the bird parasite, but there seems to be no reason for regarding it as belonging to a genus distinct from that of the human parasites.

P. præcox is common in birds of tropical and subtropical countries, but is also found, though to a less extent, in more temperate climates. It has been described from England, France, Germany, Austria, Switzerland, Italy, and South Russia. It occurs also in North America, Japan, Sumatra, and Australia, and is common in India, Africa, and other tropical countries where birds have been examined. In no place, however, is it such a common parasite as halteridium. It is most usually seen in small birds, such as the sparrow, finch, and lark, but it also occurs in larger

birds, like the pigeon, crow, owl, partridge, duck, domestic fowl, guinea-fowl, and others.

In certain cases distinct names have been given. Johnston and Cleland (1909a) gave the name *P. passeris* to a form seen by them in *Passer domesticus* of Australia, while Novy and McNeal (1904) named the parasite seen by them in *Merula migratoria* of North America *P. vaughani*. The name *P. wasielewskii* was suggested by Brumpt (1910a) for the form seen in the little owl, *Athene noctua*, which, though morphologically identical with *P. præcox*, is not inoculable to canaries. *P. bizuræ* is the name given by Gilruth, Sweet and Dodd (1910) to the parasite of the Australian bird (*Bizura lobata*), while Laveran and Marullaz (1914) described as *P. tenue* a small parasite of the Japanese bird *Liothrix luteus*. It seems very probable that several distinct species occur in birds, but the parasites have not as yet been sufficiently studied to justify the separation of species.

Plasmodium præcox (Grassi and Feletti, 1890).—As with other members of the genus *Plasmodium*, the whole of the vertebrate cycle of this hæmosporidian takes place in the red blood-corpuscles. It is very similar to that of the human parasites, the difference which occurs being due largely to the fact that the red blood-corpuscles of birds are nucleated (Plate VI., 1-10, p. 882). The youngest forms are seen as tiny cytoplasmic structures in the cells, and when stained by Romanowsky stain, show a blue cytoplasm and red chromatin dot or nucleus. A vacuole is soon formed, and the parasite resembles the small rings of *P. falciparum*. As growth proceeds, dark pigment granules appear, and the host cell becomes distorted, while its nucleus is displaced to one side or end of the cell, or even out of the cell. In this latter respect *P. præcox* differs from halteridium, which develops in the red cell without deforming it or displacing the nucleus to any extent. The fully-formed schizont varies considerably in size, and there is much doubt as to the period of growth. It is stated to be as long as four or five days, but the irregularity of the infection and the fact that at any one moment parasites of all stages of development can be seen in the blood, would suggest that the growth period may vary. The small schizonts, which produce only about six merozoites, may have occupied a shorter time to reach maturity than the large schizonts, which produce from sixteen to twenty-four merozoites. The small schizonts have a diameter of about 4 to 5 microns. while the largest ones occupy about half the red cell, the nucleus of which is displaced into the other half, the red cell itself being altered in shape and pale in colour. Schizonts intermediate in size occur between these extremes. Sometimes two or more schizonts occur in a single cell, and it is probable that the large schizonts, which have been described as producing as many as thirty-six merozoites, are in reality two closely applied parasites. In the mature

schizont, the pigment is usually collected into a single dark mass. The gametocytes are slightly elongate ovoid bodies about the size of the largest schizonts (Plate VI., 9-10, p. 882). They are often broader at one end than the other, and have irregularly distributed pigment granules. The female stains more deeply and has a more compact nucleus than the more faintly staining male.

The infection in naturally infected birds is usually a light one, but in artificial infections, which are readily produced by inoculation of blood, it may be very heavy, practically every red blood-corpuscle harbouring one or more parasites. In such cases a single field of a blood-film may show every form and stage of growth of the parasite. In birds like the canary these intense infections are frequently fatal, and *post-mortem* there is found to be a marked hypertrophy of the liver and spleen, which are very dark in colour. The heart is also enlarged, and the bone marrow pale. Sections of the organs show a distribution of pigment similar to that found in human malaria. In experimentally infected birds which survive, the height of development is reached in eight to ten days, after which the parasites become reduced in number and gradually disappear completely. There is very little tendency to relapse, as occurs so commonly in human malaria. That relapses do occur has been demonstrated by Ben-Harel (1923), who attributes them to a sudden revival in the rate of multiplication, which has been continuous throughout the infection, and not to the intervention of parthenogenesis or any other peculiarity. Laveran and Lucet (1905) attributed to this parasite a fatal epidemic which occurred amongst partridges imported to France from Hungary.

In nature the infection is transmitted from bird to bird by mosquitoes of the *Culex* type. The mosquito with which Ross (1898b) first succeeded in demonstrating the cycle was probably *C. fatigans*, and it was with this mosquito that Daniels (1899) confirmed Ross's observations in India (Figs. 396, 397). Koch (1899a), working in Italy, obtained development in *C. nemorosus* (*Aëdes nemorosus*). and Grassi in *C. pipiens*. Sergent, Ed. and Et. (1907), and Neumann (1908a) demonstrated that the complete cycle could take place in *Aëdes argenteus* (*Stegomyia fasciata*), but only 11·4 per cent. of the mosquitoes actually fed became infected. In the case of *C. pipiens* and *C. fatigans* the great majority became infected. The development in *A. argenteus* proceeded more slowly than in *Culex*. Sergent, Ed. and Et. (1918), showed that a complete development could occur in *Theobaldia longiareolata*, *Aëdes mariæ*, and *Culex hortensis*.

The method of development is identical with that of the human parasites in anophelines, the time occupied for the completion of the cycle varying with the temperature. Working with what was probably *C. fatigans* in Bagdad during the summer, the writer obtained complete

development, including infection of the salivary glands, in less than five days. Sergent, Et. (1919), carried out experiments on the development of *P. præcox* in *C. pipiens* under varying conditions of temperature. He found that the optimum temperature lay between 20° and 30° C. Mosquitoes exposed to 12° C. for the first six hours after the infecting feed and then transferred to favourable temperatures rarely became infected. With exposure for periods longer than six hours the number of mosquitoes subsequently showing infections diminished till after an exposure of eight days none became infected. If, after feeding, mosquitoes were kept at a temperature varying between 17·5° and 24° C., development took place very slowly, sporozoites not appearing in the salivary glands till after the expiry of two months. Sporozoites which have already developed in the salivary glands cease to be infective if the mosquitoes are kept for five months at a temperature varying from 8° to 25° C.

The number of oöcysts varies with the number of gametocytes in the blood. In some cases enormous infections of the mosquito's stomach occur, and in one instance the writer noted that the whole stomach wall appeared to be replaced by oöcysts, which not only formed a continuous layer on its outer surface, but also occurred in the wall itself, the cellular elements of which were reduced to a mere network. In other cases only a small number or even a single oöcyst is found. As already remarked, the development in the mosquito is exactly comparable with that of the human parasites in anophelines, and calls for no further description (Fig. 407).

Birds which have recently recovered from an infection as judged by blood examination are relatively immune to further inoculation, but this resistance quickly passes off, so much so that it is supposed that in the resistant birds the infection is still present in a mild degree. Sergent, Et. and Ed. (1910), claimed to have immunized canaries against infection by injecting them with the killed sporozoites obtained from mosquitoes, while Sergent, Et., and Hempl (1917) showed that of five canaries which had recovered from an infection two and a half years before, only one was susceptible to reinoculation. Whitmore (1918), on the other hand, believes that no immunity exists after the parasites have completely disappeared. He was led to this view by the fact that bird's blood may be still infective to other birds, even when microscopic examination failed to reveal the parasite. Sergent, Et. and Ed. (1910, 1921, 1921*b*), have studied bird malaria in reference to immunity and treatment. They have used strains which were virulent for canaries, in which a relatively high percentage of fatal infections occurs after inoculation. They (1921) have noted that injection of old sporozoites which have remained in the

salivary glands of mosquitoes (*C. pipiens*) for some months or sporozoites which have been kept some time *in vitro* protects birds to the extent of decreasing the mortality rate. Similarly the inoculation of birds with blood drawn during the incubation period before parasites have appeared modifies considerably a subsequent inoculation as regards the acuteness of the infection.

That birds can be readily infected by inoculation of blood containing parasites was first demonstrated by Celli and Sanfelice (1891), who transmitted the disease from lark to lark. This experiment was repeated by Grassi and Feletti (1891) and Laveran (1891), while Ziemann (1898) inoculated the parasite found in the greenfinch into other greenfinches.
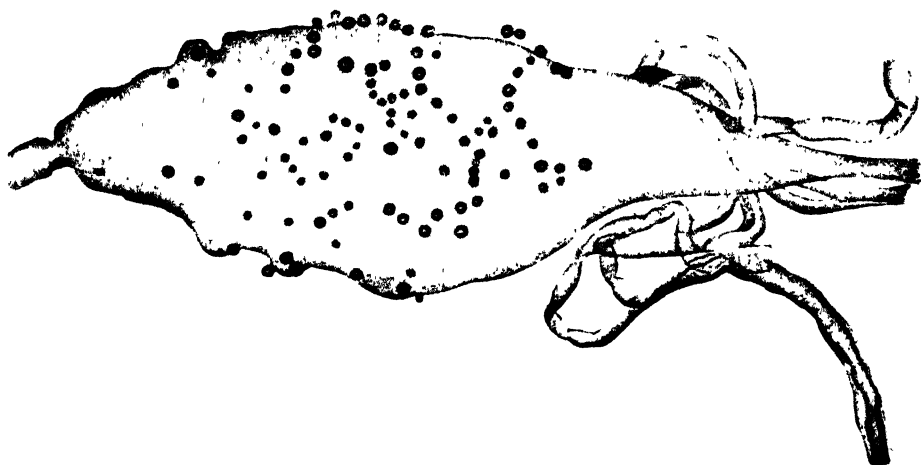
FIG. 407. – STOMACH OF *Culex fatigans*, SHOWING NUMEROUS OÖCYSTS OF *Plasmodium præcox* (× 35). (ORIGINAL.)

From preparation made by fixing stomach between slide and cover-glass with Schaudinn's fluid and staining with hæmatoxylin.

Koch (1899, 1899a) showed that the naturally occurring parasite of the sparrow and goldfinch was inoculable to sparrows, canaries, goldfinches, crossbills, and robins. Ruge (1901) inoculated sparrows and canaries with a strain from the sparrow. Wasielewski (1908), obtaining a strain from the chaffinch, infected other chaffinches as well as the mountain finch, greenfinch, canary, lark, and goldfinch. He also passed the naturally occurring parasites of the sparrow, goldfinch, and yellow-hammer to canaries, as well as that of the goldfinch to linnets. Ross (1898b) had already shown that mosquitoes fed on sparrows were able to infect crows and larks, so that it is evident that one and the same species of *Plasmodium* is able to infect a number of different birds.

PLASMODIA OF LIZARDS.

A number of pigmented parasites has been described from the blood of cold-blooded animals (Plate XVII., 6-30, p. 982). Some of these belong to the genus *Plasmodium*, as far as can be judged from a knowledge of the vertebrate forms only, in that the schizogony cycle and production of gametocytes takes place in the red blood-corpuscles.

Plasmodium agamæ (Wenyon, 1909).—This pigmented parasite was first seen by the writer (1909) in the red blood-corpuscles of the lizard, *Agama colonorum*, of the Southern Sudan (Plate XVII., 16-20, p. 982). It was placed in the genus *Hæmoproteus*, but as schizonts occur in the red blood-corpuscles, it is evidently a member of the genus *Plasmodium*, as pointed out by the writer (1915). The youngest stages are minute bodies, consisting of blue staining cytoplasm and a chromatin dot. They occur at the ends of the red cells. The fully-formed schizonts are 4 to 7 microns in diameter and possess up to six nuclei. Male and female gametocytes are elongate pigmented bodies measuring about 14 by 4 microns, and they lie at the side of the nucleus in the halteridium manner. The red cells, which normally measure 13 to 18 by 8 to 12 microns, are not distorted or enlarged by the parasite. Flagellation of the male gametocyte, with production of male gametes, was observed. The parasite has also been seen in Nigeria by Macfie (1914a), who called it *Hæmocystidium agamæ*, and by Adler (1924a) in West Africa. who placed it in the genus *Plasmodium*. Adler found that the schizonts in the red blood-corpuscles gave rise to as many as seventy merozoites.

Plasmodium mabuiæ Wenyon, 1909.—Like the last described species, this organism is a parasite of a lizard (*Mabuia quinquetæniata*) of the Southern Sudan (Plate XVII., 21-25, p. 982). The youngest stages are small, irregular, amœboid masses of cytoplasm which are closely applied to the nuclei of the host cells, and from which they thrust out pseudopodia into the cytoplasm of the cell. Pigment is present in the form of fine grains, which may be clustered into several groups. The larger schizonts are rounded bodies separate from the host cell nucleus. They have a diameter of about 5 microns, and contain up to six chromatin dots. Male and female gametocytes measure about 8·5 by 5·5 microns. The red cells of the host, which measure 15 to 20 by 6 to 10 microns, are not deformed by the parasite.

Plasmodium tropiduri Aragão and Neiva, 1909.—This form is a parasite of the Brazilian lizard, *Tropidurus torquatus*. The usual young and growing forms occur. The mature schizonts measure 7 to 8 microns in diameter, and produce up to twelve merozoites. Male and female gametocytes 6 to 9 microns in diameter occur. A slight deformity of the end of the
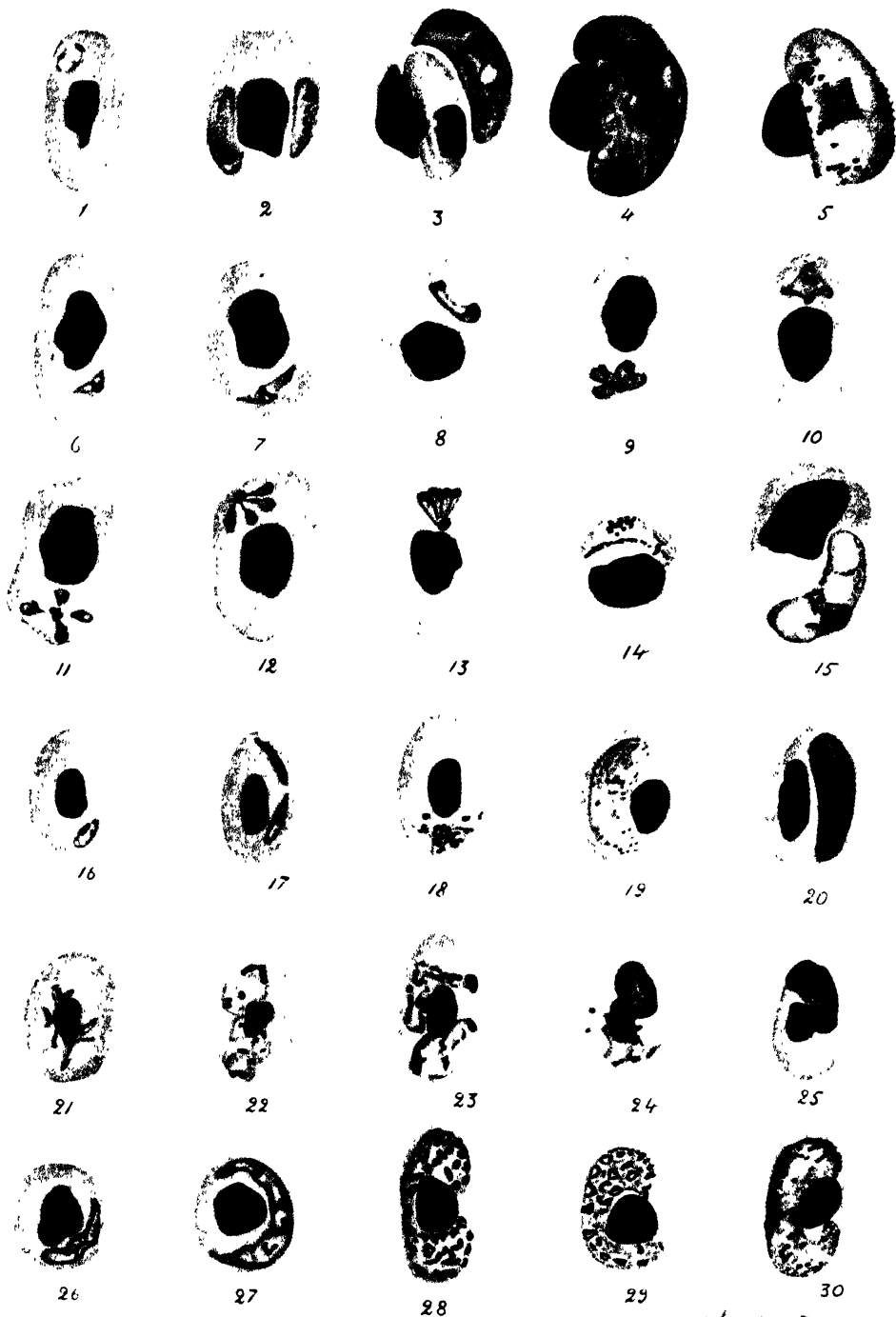
Hæmosporidia of reptiles.—1-5. *Hæmoproteus mesnili* of African cobras (*Naja* and *Sepedon*).   6-15. *Plasmodium minasense*.   16-20. *Plasmodium agamae*.   21-25. *Plasmodium mabuiae*.   26-30. *Plasmodium diploglossi*.

red cell in which the parasite is found takes place. The close resemblance of the parasite to *P. præcox* of birds is noted.

Plasmodium diploglossi Aragão and Neiva, 1909.—The parasite occurs in the red blood-corpuscles of the snake lizard, *Diploglossus fasciatus*, of Rio de Janeiro (Plate XVII., 25-30, p. 982). Unsuccessful attempts were made by its discoverers to inoculate other lizards (*Tropidurus torquatus, Hemidactylus mabuia, Ameiva surinamensis*, and *Mabuia agilis*). The youngest forms of the parasites are minute ring forms not more than 2 microns in diameter. As it increases in size, the parasite passes round the nucleus of the host cell till it is completely surrounded. Nuclear multiplication takes place till as many as forty nuclei are present. The gametocytes are similar in shape to the adult schizonts. The normal red cell measures 15 by 9 microns, but with growth of the parasite it becomes pale in colour and enlarged till it measures 19 by 11 microns. The cell, however, is not distorted, nor is its nucleus displaced.

Plasmodium minasense Carini and Rudolph, 1912.—This organism was first seen in the Brazilian lizard, *Mabuia agilis*, in 1912. A form, apparently identical from a morphological standpoint, was described by the writer (1915) from the iguana (*Iguana sapidissima*) of Trinidad (Plate XVII., 6-15, p. 982). The schizonts are 4 to 5 microns in diameter and produce four merozoites, which are arranged either as a cross with the pigment at the centre, or as a fan or cone with the pigment at the apex. The gametocytes are spherical or ovoid, or more elongate, in which case they lie round the nucleus. They may reach a length of 8 to 9 microns, with a breadth of 2 to 4 microns. The pigment in all forms is very scanty. Leger, M., and Mouzels (1917) described as *P. carinii* what may be the same species from *I. nudicollis* of Guiana. The resemblance of this parasite to *Babesia quadrigemina* is very striking (Fig. 426).

ACTION OF DRUGS ON MALARIAL PARASITES.

As malaria is such an important and widespread disease of man, numerous drugs have been investigated with a view to the discovery of a cure. Though arsenic, especially in organic compounds, has a definite action on the parasites, particularly *P. vivax*, the only drugs which can be regarded as specifics are the alkaloids which can be extracted from the bark of cinchona plants. Of these quinine holds first place, though others, such as cinchonine, quinidine, cinchonidine, and quinoidine, are not without action on the malarial parasites. It is known that quinine is highly toxic to free-living protozoa, and it was naturally imagined that the drug, when introduced into the body, acted directly on the malarial parasites themselves. Many authorities, however, believe that the process is not so

simple, and that quinine acts indirectly by stimulating the cells of the body to produce substances harmful to the parasites.   Morgenroth (1918) believes that the quinine actually combines with the red blood-corpuscles, and that the merozoites are thereby prevented from entering them, so that they fall a prey to the leucocytes or other destructive agents in the blood.   Quinine in the form of its salts in comparatively small doses, such as 10 grains or even less, is capable in many cases of causing rapid disappearance of the asexual forms of malarial parasites from the blood. But, as occurs in the treatment of so many protozoal infections, it is known that, though the vast majority of the parasites are destroyed, some survive, either because they are peculiarly tolerant of the drug, or are situated in places to which the drug cannot gain access.   It has been suggested that they may escape its action by inclusion in cells other than the red blood-corpuscles.   Whatever may be the explanation, those parasites which survive commence multiplying again, and infections as intense as those previously present may supervene.   By continued action of the drug this reproduction is frequently kept in abeyance, and actual attacks of fever or relapse may be prevented.   It has been suggested that the continued use of quinine will produce quinine-fast strains of malarial parasites comparable with the arsenic- and antimony-fast strains of trypanosomes, but the evidence for this is not conclusive.   Weygandt and Mühlens (1920) have noted that quinine quickly gets rid of a malarial infection in an individual who has been inoculated from a quinine-resistant malarial case.   Such an observation suggests that the resistance to quinine is not due to the parasite, but to some peculiarity of the particular individual infected.   It must be remembered that infections may disappear as a result of the natural protective processes of the body, and in many cases it seems that quinine acts by keeping the number of the parasites at a minimum till cure is effected in a natural manner.   Quinine is frequently taken as a prophylactic during exposure to bites from infected mosquitoes. but much difference of opinion as to its value exists.   It has been generally assumed that if quinine occurs in the blood and body fluids when a mosquito injects sporozoites, these are killed by the drug.   Though there is no evidence to support this view, it still appears to be a fact that, unless exposure to infection is very great or extends over long periods, small doses of quinine taken as a prophylactic will, in some cases, prevent actual malarial attacks in the individuals exposed.   It does not necessarily follow from this that the sporozoites are killed, for it is just as reasonable to suppose that the drug is acting on the earliest intracellular phases in the human body after schizogony has commenced.   Observations carried out by York and Macfie (1924a) during the infection of cases of general paralysis are in agreement with this view.   They have shown that 5 grains

of quinine *per diem*, if taken before and for a few days after an infected mosquito bite, will not prevent infection, but that if it be continued beyond the period when the first symptoms would be expected to appear it will ward off the attack.

In connection with the culture of human malarial parasites, reference has been made above to the difficulty of succeeding if this is attempted in the case of patients who are undergoing quinine treatment. It has also been noted that the gametocytes of *P. vivax*, as regards their capacity for development in mosquitoes, are much more susceptible to the action of quinine than those of *P. falciparum*.

Quinine has generally been supposed to have little or no effect on the malarial parasites of animals. Leger, M., and Bouilliez (1913) found that it did not affect *P. inui* in monkeys, though Gonder and Rodenwaldt (1910) had noted that the drug definitely controlled infections due to *P. kochi* in these animals. Many observations have been made on the influence of quinine on *P. præcox* of bird malaria. Employing a strain of this parasite, which proved fatal to canaries in a percentage of 30 to 60, Sergent, Et. and Ed. (1921c), noted that the daily intramuscular administration of 2 to 4 milligrams of quinine hydrochloride was capable of preventing the appearance of the acute and heavy fatal infections. Though under these conditions parasites could not be found in the peripheral blood by microscopic examination, that they were still present was proved either by inoculation of other birds or the infection of mosquitoes feeding on them. The prophylactic action of quinine was also tested, and in this case, again, though the drug prevented the occurrence of acute fatal infections, parasites nevertheless established themselves in the body. These were proved to be still present after many months of treatment. The parasites which persisted gave rise to a milder infection when inoculated to other canaries, but after two or three passages the strain acquired its original virulence. Experiments with arsenobenzol, tartar emetic, and other drugs showed that if these had any action at all, it was inferior to that of quinine.

3. *Sub-Order:* Piroplasmidea.

In this sub-order are included certain parasites which inhabit red blood-corpuscles of mammals, but do not form the pigment (hæmozoin) characteristic of members of the genera *Hæmoproteus* and *Plasmodium* (Plate XVIII., p. 986). Each parasite consists of a minute portion of cytoplasm and a nucleus, and in dry films stained by Romanowsky stains it appears as a blue staining cytoplasm and a red staining chromatin portion, consisting frequently of a red granule with a string of finer granules extending from it. When a vacuole is present in the cytoplasm, as is not

infrequently the case, it may be difficult to distinguish from a young ring form of a malarial parasite. Whereas the majority of the malarial parasites reproduce by a process of schizogony and give rise to a fairly large number of merozoites, the piroplasmata reproduce by a division into only two or four daughter individuals. However, certain members of the genus *Plasmodium*, as, for instance, *P. minasense* of lizards (Plate XVII., 6-15 p. 982), divide into, or rather bud off, four merozoites only, and were it not for the pigment which is present, it would be impossible to distinguish these from certain piroplasmata, such as *Babesia quadrigemina* (Fig. 426) and *Achromaticus vesperuginis* (Plate XVI., 28-32, p. 974), which reproduce in a similar manner. These forms appear to stand as a connecting link between the Hæmosporidiidea and the Piroplasmidea.

A parasite of cattle (*Theileria parva*), the cause of East Coast fever, differs from other piroplasmata in that a definite schizogony process occurs, not in the red blood-corpuscles, but in the internal organs within cells which are probably endothelial in nature (Fig. 428). The schizonts (Koch's blue bodies) produce a large number of merozoites, and on this account it is impossible to group this form with the members of the genus *Babesia*, which reproduce only in the red blood-corpuscles (Plate XVIII., 31-38, p. 986).

Smith and Kilborne (1893) published an account of the transmission by ticks of *Babesia bigemina*, the cause of Texas or red-water fever of cattle. They not only made the remarkable discovery of the possibility of transmission of protozoal parasites by arthropod hosts, but also noted that the virus passed through the egg of the tick, thus causing the succeeding generation which hatched from the eggs to be infective. Though the method of transmission was thus discovered, the actual development of the parasites in the body of the invertebrate has not been traced. On this account it is not possible to assert that gametocytes are present in the red blood-corpuscles of the vertebrate host, though this is probable. If it be assumed that this is the case, then the grouping of the piroplasmata in the two families, Babesiidæ and Theileriidæ, corresponds with the subdivision of the pigmented hæmosporidia into the two families, Plasmodiidæ and Hæmoproteidæ. In the one the red cells contain asexually reproducing forms and gametocytes, while in the other the red cells contain only gametocytes, the schizonts occurring in the endothelial cells of the vessels.

Some piroplasmata, as, for instance, *Babesia bigemina* of cattle and *B. canis* of dogs, are relatively large, whereas others, such as *B. mutans* of cattle, are very small, so much so that there is great difficulty in making out the details of their structure. The larger forms, if round, may have a diameter of 3 or even 4 microns, or, if elongated, they may extend from one side of the red corpuscle to the other. The smaller forms may be

PLATE XVIII.
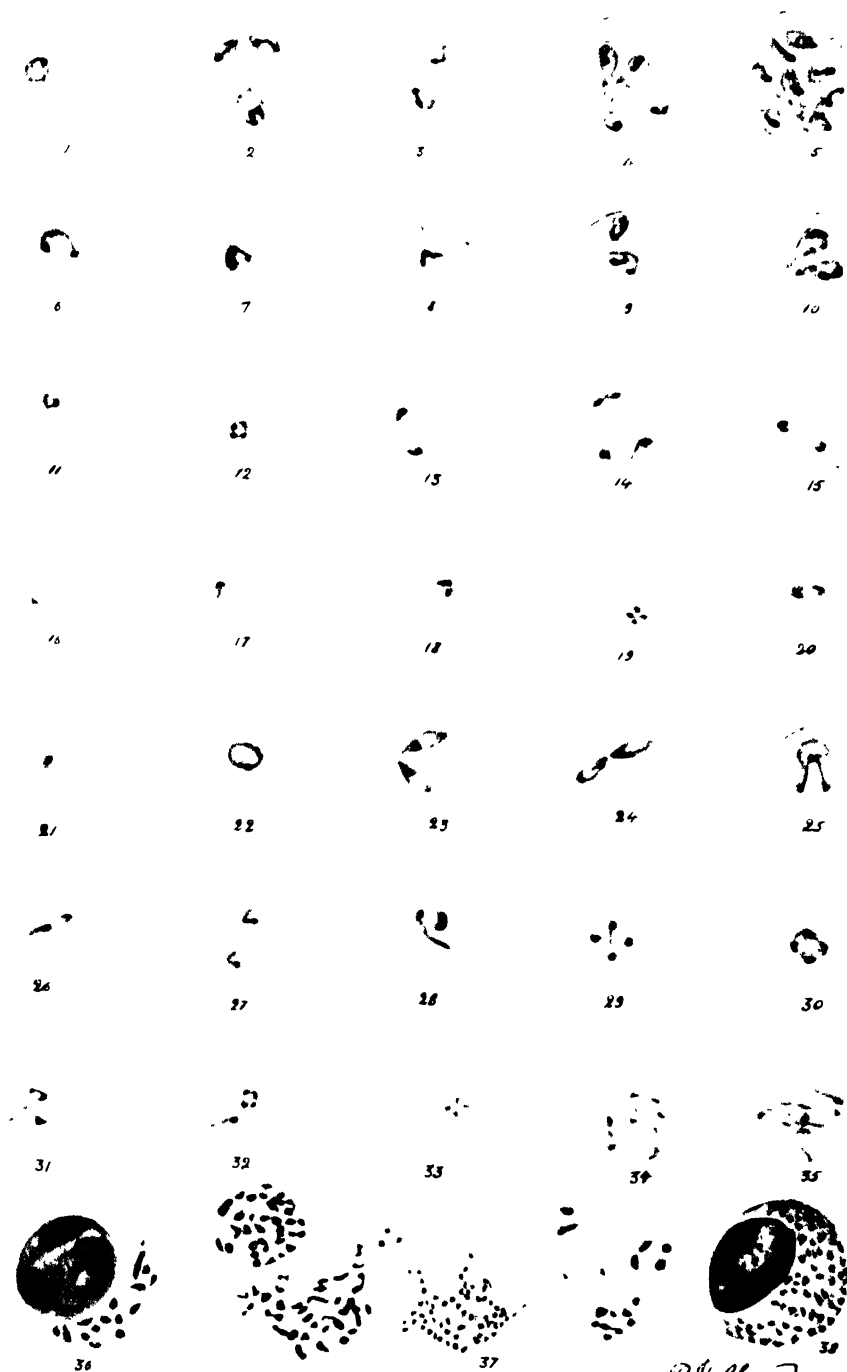
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barely 1 micron in diameter. In some of the latter, the cytoplasmic portion is much reduced or may appear to be absent, so that they approach in appearance the anaplasmata, which occur in the red cells as red staining granules (Fig. 438).

It has been clearly demonstrated that cattle are liable to infection with at least three species of *Babesia* (*B. bigemina*, *B. bovis*, and *B. mutans*), in addition to *Theileria parva*. Parasites like *B. bigemina*, which causes red-water fever in cattle, were noted by Guglielmi (1899) and Laveran (1901) to give rise to a similar disease in horses. Dreyer (1910) seems to have been the first to realize that two types of infection occurred in horses, the one producing red-water and the other not. The parasite occurring in the latter condition was studied by Laveran (1901), who named it *Piroplasma equi*. It is a smaller parasite, and differs morphologically from that producing red-water fever, which was named *P. caballi* by Nuttall and Strickland (1910, 1912). They proved by experiments that an animal which has recovered from an infection with one of these parasites was not reinoculable with the same parasite, but succumbed to an infection with the other. Similar experiments were conducted with like results by du Toit (1919) in Italy. From the work of Lestoquard (1924, 1925) it appears that sheep and goats are liable to infection with a series of parasites which correspond very closely with those of cattle. Many other forms have been described, but in most cases very little is known about their structure and life-history.

The first piroplasm to be discovered was the one observed by Babes (1888) in the red blood-corpuscles of Roumanian cattle. He stated that it resembled a *Gonococcus*. Later (1889) he referred to it as *Hæmatococcus*, and again (1890a) as " microcoque de l'hémoglobinurie ou *hematococcus.*" He appeared to have had no intention of naming the organism. and used the term *Hæmatococcus* as meaning a coccus of the blood. In any case, the name *Hæmatococcus* had been previously used for a flagellate (Fig. 131). Babes (1890) claimed to have isolated the organism in culture, but he was confusing it with bacteria which grew in the media used. Babes (1892) again refers to "l'hématococcus de l'hémoglobinurie du bœuf," and describes as "l'hématococcus du mouton" a similar parasite of sheep. Smith and Kilborne (1893) then gave the name *Pyrosoma bigeminum* to the parasite of Texas fever of cattle, overlooking the fact that the name *Pyrosoma* had been given previously to an Ascidian. Starcovici (1893) discussed the nature of these parasites, and gave to the forms discovered by Babes the names *Babesia bovis* and *B. ovis*. Wandolleck (1895), objecting to the name *Pyrosoma*, not because of its previous use, but because its derivation does not signify " pear-shaped," as Smith and Kilborne intended, proposed the generic title *Apiosoma*, while Patton, W. H. (1895),

suggested the name *Piroplasma*, which has been in use for a number of years. There can be no doubt, however, that Starcovici's name *Babesia* is the correct one for these parasites, and that he was the first actually to name the organisms studied by Babes.

Parasites very similar to those producing red-water fever in cattle were described by Babes (1892) from sheep, by Piana and Galli-Valerio (1895) from dogs, and by Guglielmi (1899) from horses. Owing to the fact, not realized by the early observers, that cattle, horses, and sheep are liable to infection with more than one species of parasite, some confusion naturally arose. Koch (1898) observed the forms now known as *Theileria parva* in cattle in Africa, and regarded them as young forms of *B. bigemina*. Later (1903), immunity experiments convinced him that two distinct parasites were involved. Theiler (1904–1907) then investigated the small parasites of cattle, and discovered that there were actually two species, one of which he named *P. mutans*, a comparatively benign organism, and the other *P. parva*, the cause of East Coast fever.

Subdivisions of the Piroplasmidea.

All the piroplasmata of cattle, horses, sheep, and other animals were at first placed in the genus *Piroplasma* (or *Babesia*). Attempts were then made to divide the group into a number of genera, but this, in the writer's opinion, has led to the utmost confusion. As regards the parasites of cattle, of which there are three well-defined species (*B. bigemina, B. bovis,* and *B. mutans*), these have been placed in three distinct genera. Bettencourt, França and Borges (1907) concluded that the small parasites which had been named *P. parvum* and *P. mutans* by Theiler belonged to a different genus, for which the name *Theileria* was proposed. This genus was based on the characters of the forms in the red blood-corpuscles, which are very similar in the two species. The former, however, reproduces by schizogony in the internal organs, and the latter, like the other members of the genus *Babesia*, in the red blood-corpuscles. Consequently, the genus *Theileria* was reserved for the form named by Theiler *P. parvum*, the cause of East Coast fever, while the other parasite was returned to the genus *Babesia*. From Babes' descriptions it appears probable that the parasite of cattle studied by him was chiefly the one of intermediate size, for which Starcovici's name *Babesia bovis* has priority. If it be accepted that all these parasites belong to one genus, then the largest form is *B. bigemina*, that of intermediate size *B. bovis,* and the smallest *B. mutans*. Du Toit (1918) believes that the large form should be placed in a distinct genus, for which he employs the name *Piroplasma*, given by Patton, W. H. (1895), to the large parasite studied by Smith and Kilborne. The small

form, again, he places in a distinct genus, for which the name *Gonderia* is proposed. Some observers believe that the generic name *Babesia* should be reserved for the largest parasite, and not for the one of intermediate size. Accordingly, Sohns (1918) proposed to place the one of intermediate size in a new genus, *Microbabesia*, while Mesnil (1919) suggested yet another name, *Babesiella*, for this parasite. According to du Toit, the parasites of cattle are *P. bigeminum*, *B. bovis*, and *G. mutans*, while other writers name them *B. bigemina*, *Babesiella bovis*, and *G. mutans*. It will thus be seen that the position regarding the correct nomenclature of the parasites of cattle is extremely complicated, and it seems to the writer that nothing is to be gained by placing them in different genera. They will be regarded as belonging to the one genus, *Babesia*, the type species of which is *B. bovis*. As regards the parasites of horses, of which there are two well-defined species (*B. caballi* and *B. equi*), França (1910) placed the small form in a distinct genus as *Nuttallia equi*, while the larger one is retained in the genus *Babesia* by some observers, though du Toit places it in the genus *Piroplasma* with the largest of the three parasites of cattle. Lestoquard (1925) has shown that sheep are liable to infection with three species, which correspond with the three forms in cattle. Placing these in different genera, he recognizes *P. ovis*, *Babesiella ovis*, and *G. ovis*. His reason for doing so appears to be chiefly a desire to bring the parasites into line with the corresponding ones of cattle, which are presumed to belong to these three genera. They will be regarded here as belonging to the one genus, *Babesia*. Nicolle (1907) discovered a parasite in the gondi in Tunis, and named it *P. quadrigemina*. Nuttall (1908) placed it in a new genus, *Nicollia*. Another form was discovered by Nuttall (1910) in the jackal (*Canis adustus*) of British East Africa, and was subsequently (1912) placed by him in a separate genus as *Rossiella rossi*. Another genus, *Smithia*, was created by França (1910) for a parasite (*S. microti*) of a vole (*Microtus incertus*) of Portugal, while Carini and Maciel (1914a) gave the name *Rangelia vitalii* to a form which was pathogenic for dogs in Brazil. These four names (*Nicollia*, *Rossiella*, *Smithia*, and *Rangelia*) will be regarded as synonyms of *Babesia*. A non-pigmented parasite of bats, discovered by Dionisi (1898), and named by him (1899) *Achromaticus vesperuginis*, probably belongs to the genus *Babesia*, though a parasite of moles, first described by Thomson, J. D. (1906), and named *Elleipsisoma thomsoni* by França (1912b), may be sufficiently distinct to justify its inclusion in a separate genus.

From the above account it will be clear that there is little agreement regarding the nomenclature of the piroplasmata. França (1917, 1918) gave a detailed account of the classification of these parasites, and recognized a number of genera. França's classification was modified by du Toit (1918)

and new genera introduced. Though the writer believes that these genera are not valid, du Toit's system will be considered, as many writers accept it either as he gave it or in a modified form. In his system du Toit correctly recognizes two families, the Babesidæ and Theileridæ, which have already been defined. They are subdivided as follows:

Family: BABESIIDÆ Poche, 1913.

1. *Genus:* **Babesia** Starcovici, 1893.—Parasites which assume the pear shape, and become arranged in couples in the cells. Reproduction is by division into two.

(*a*) *Sub-Genus:* **Babesia** (Starcovici, 1893).—Small forms under 2·5 microns in length. They are mostly of irregular form, though the characteristic pear-shaped individuals sometimes occur. Multiplication is by a simple division into two. Includes *B. bovis* (*B. divergens*), *B. ovis, B. argentina, B. muris, B. gibsoni, B. soricis.* (For these forms Sohns (1918) suggested the generic name *Microbabesia,* and Mesnil (1919) the name *Babesiella.*)

(*b*) *Sub-Genus:* **Piroplasma** (Patton, 1895).— Larger parasites over 3 microns in length, and containing two nuclei, one large and the other small. The typical form is the pear-shaped couple. Division into two takes place by a budding process. Includes *P. bigeminum, P. canis, P. pitheci, P. avicularis, P. leporis, P. caballi, P. trautmanni.*

2. *Genus:* **Nicollia** Nuttall, 1908.—Parasites which are ovoid or pear-shaped and have two nuclei. Division into four, producing a four-leafed arrangement in the cell. Includes *Nicollia quadrigemina.*

3. *Genus:* **Nuttallia** França, 1910.—Parasites which are ovoid or pear-shaped. Division into four, producing the cross arrangement. No typical rod forms. Includes *N. equi, N. herpestidis, N. ninensis, N. asini, N. muris, N. microti,* and unnamed species from Transcaucasian sheep and horses and *Cervus aristotelis.*

4. *Genus:* **Smithia** França, 1910.—Parasites which become pear-shaped and stretch completely across the red cell. There is no tendency to arrangement in couples. Division in the cross form. Includes *S. microti* and *S. talpæ.*

5. *Genus:* **Rossiella** Nuttall, 1912.—Parasites of large size and rounded form. One to four in each red cell. The nucleus is large and round. Division by fission into two. Includes *R. rossi.*

6. *Genus:* **Gonderia** du Toit, 1918. — Parasites of small size and rounded form. Division into four in the cross arrangement. The daughter forms are composed mostly of chromatin. Includes *G. mutans, G. dama, G. hirci, G. cellii, G. hippotragi, G. buffeli, G. stordii, G. brimonti,* and unnamed species from *Cephalopus grimmi, Rangifer tarandus, Gazella* sp., and Congo sheep.

1. *Genus:* **Theileria** Bettencourt, França and Borges, 1907.—Parasites of a round or rod form. Multiplication by division into two in the red cells or by schizogony in cells of the lymphatic system. Includes *T. parva, T. annulata, T. ovis, T. tachyglossi,* and an unnamed form from the eland. (There is no real evidence that multiplication in the red cells occurs.)

2. *Genus:* **Rangelia** Carini and Maciel, 1914.—Parasites which are round, oval, or pear-shaped. Multiplication by division into two in the red cells or by schizogony in the endothelial cells. Includes *R. vitalii.*

The parasites included in the genera mentioned above agree with one another in that they consist of cytoplasm and chromatin, and are devoid of pigment, and it seems to the writer that there are insufficient grounds for the recognition of numerous genera, which are based chiefly on difference in size and shape, and on the method of division into two or four daughter individuals, and the arrangement and position of these in the cell. The genus *Plasmodium* includes forms which produce sixteen, eight, or only four merozoites. These are arranged either irregularly, in definite rosettes, or in a fan-like manner within the red cell. The parasites may be large, as in the case of *P. vivax,* or small, as in *P. falciparum.* If the various non-pigmented parasites are divided into different genera according to these characters, the genus *Plasmodium* itself would have to be similarly split up. It seems that there is nothing to be gained by such subdivisions, especially when it is realized that in no single instance is the complete cycle in the invertebrate known. In du Toit's classification, the three parasites of cattle are placed in different genera under the names *Piroplasma bigeminum, Babesia bovis,* and *Gonderia mutans.* The chief distinction between these is size. In *B. bovis* the arrangement of pear-shaped forms in couples is said to occur rarely, a feature which is given as a generic character. It is further stated that this parasite reproduces by simple division into two, instead of by the budding process seen in *P. bigeminum.* From what the writer has seen, *B. bovis* multiplies in the same way as *P. bigeminum.* *G. mutans* is said to be round in form and to divide into four, but pear-shaped parasites are also found, while division into two as well as four occurs. It seems, therefore, impossible to name any definite characters which will separate these three genera, and the same remark applies to the others. The sub-order Piroplasmidea will, therefore, be considered as comprising two families and two genera only.

1. *Family:* BABESIIDÆ Poche, 1918.— Parasites which multiply in the red blood-corpuscles by division into two or four. They are of

varying size and shape, and have a tendency to arrangement in couples of pear-shaped individuals. The forms in the corpuscles are asexually reproducing individuals, and possibly gametocytes.

Genus: **Babesia** Starcovici, 1893.

2. *Family:* THEILERIIDÆ.—Parasites which multiply by schizogony in endothelial cells of the vessels, and finally invade the red cells, within which they occur as round, ovoid, rod-like, or irregular forms. They show no tendency towards a paired arrangement. The forms in the red blood-corpuscles do not reproduce, and are possibly gametocytes.

Genus: **Theileria** Bettencourt, França, and Borges, 1907.

1. *Family:* BABESIIDÆ Poche, 1913.

In this family are included the non-pigmented parasites of the red blood-corpuscles of mammals, which reproduce within these cells by division into two or four daughter individuals. It is probable that certain forms are gametocytes. The species which have been properly studied can all be placed in the genus *Babesia*, which has the characters of the family.

BABESIA OF CATTLE.

A number of different species of piroplasmata have been described from cattle. The fact that the dimensions of any individual species may vary shows that size alone is of little value in the separation of species. Brumpt (1920) pointed out that a single species varies to a certain extent as it passes from one animal to another, while Rosenbusch and Gonzalez (1925) have noted in Brazil that races of *B. bigemina* from different localities vary morphologically.

It has been shown, however, by Theiler, M'Fadyean and Stockman, Lignières and Brumpt that animals which have recovered from an infection with one species may be infected by direct inoculation of blood or by the tick with another species. It has already been pointed out that such immunity tests are unreliable as a means of separating trypanosomes, and undoubtedly the same fallacies occur in the case of the piroplasmata. Immunity tests will separate distinct species, but different races of a single species may give similar results. This appears to have been clearly demonstrated by Rosenbusch and Gonzalez (1925) in Brazil. They studied the disease of cattle known as tristeza, which is caused by a parasite corresponding with *B. bigemina*. It was found that not only did the parasites from different localities vary morphologically, but that cattle which had recovered from an acute infection due to a strain from one locality were immune to this particular strain, but could be infected with strains from other localities. As will be shown below, *B. canis* of dogs of

French origin will not protect these animals against the North African virus, with which it is morphologically identical, so that if the immunity reactions are relied upon as a test of species amongst the piroplasmata, then morphologically identical forms will soon be split up into a multiplicity of species, as has occurred in the case of the trypanosomes.

As noted above, there are three fairly well-defined species of *Babesia* in cattle—viz., *B. bigemina*, *B. bovis*, and *B. mutans* (Plate XVIII., 6-20, p. 986). *B. bigemina* is a comparatively large organism, and rounded forms may have a diameter of half that of the red cell, while the elongate and pear-shaped forms are longer than this. *B. mutans* is a minute parasite, which is usually about ·5 to 1 micron in diameter, while *B. bovis* occupies an intermediate position.

Babesia bigemina (Smith and Kilborne, 1893).—This parasite was first observed by Babes (1888) in what Brumpt believes to be a mixed infection with *B. bovis*. The organism was first accurately studied in America by Smith and Kilborne (1893). who made the remarkable discovery of its transmission by ticks, an observation which demonstrated for the first time the rôle of arthropods in the carriage of protozoal diseases. As the tick in question (*Margaropus annulatus*) remained fixed to one host during all its stages of development, and only dropped off at the end of its life to lay eggs, it was evident, as they demonstrated, that the infection passed from the parent tick to the offspring through the egg.

Morphology.—*B. bigemina* is the largest of the piroplasmata of cattle (Plate XVIII., 6-10, p. 986. and Fig. 408). Round, oval, irregular, and pear-shaped forms occur. The arrangement of pear-shaped individuals in couples is characteristic of this species, as it is of the others, but the individuals of each pair lie more closely together. and do not show the same divergence of the blunt extremities. The round forms have a diameter of 2 to 3 microns, while the elongate ones may have a length of 4 microns, and extend across the diameter of the red blood-corpuscle, which in cattle measures from 5 to 6 microns. Occasionally, instead of two, four pear-shaped forms are arranged in a fan-like manner. Multiplication takes place by a characteristic budding process. as was described by Nuttall and Graham-Smith (1908) for this and other species. The nucleus, of a rounded form as seen in dried films. is described as consisting of a granule of chromatin and a string of finer granules extending from it. This string of fine granules increases in length at the expense of the large granule, which itself increases in size. Finally, the string of granules bifurcates, and the extremities of the limbs thus formed extend into two small buds of cytoplasm which appear on the surface of the parasite (Fig. 408). These buds increase in size. become pear-shaped, and absorb the cytoplasm from the body of the parasite. Eventually there result two pear-shaped individuals attached by their pointed

extremities to a small portion of cytoplasm, which is all that is left of the parent. In this the granule of chromatin is still present, and from it there extends a string of granules into each daughter pear-shaped form. The chromatin granule then divides, and by absorption of the remaining cytoplasm two separate pear-shaped parasites result. In each of these there is a chromatin granule near the pointed end, while a string of granules extends towards the blunter end. Occasionally four instead of two buds are formed in a similar manner and four daughter forms arise. These pear-shaped parasites may apparently leave the red cell and enter others, where they become more or less rounded forms which, after some increase in size, during which they may exhibit amœboid movements,
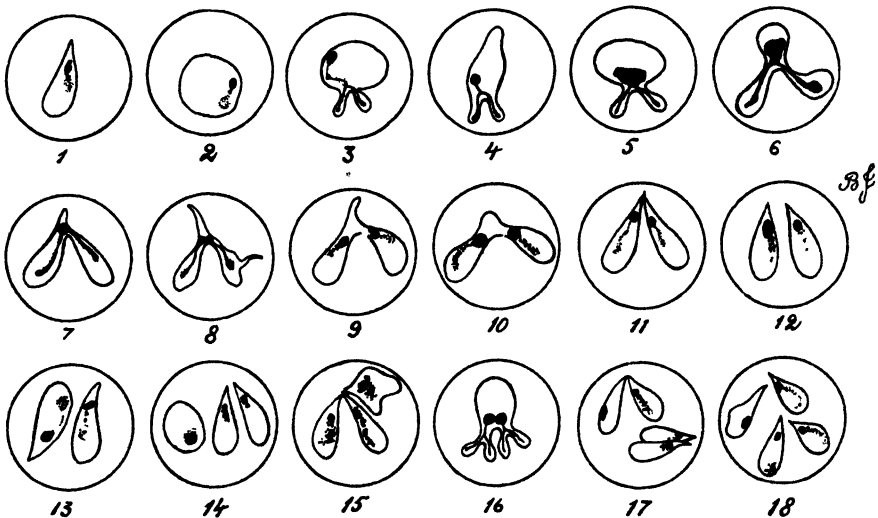
FIG. 408.—*Babesia bigemina* OF CATTLE: METHOD OF MULTIPLICATION BY BUD-DING IN RED BLOOD-CORPUSCLES (× *ca.* 3,000). (AFTER NUTTALL AND GRAHAM-SMITH, 1908.)

commence to divide again by the peculiar budding process. After their formation the pairs of pear-shaped individuals remain united for some time by their pointed extremities, which are often drawn out into fine filaments, at the point of union of which a small deeply-staining granule may occur. The details of nuclear division and bud formation in this and other species of *Babesia* have only been studied in dried films, so that little reliance can be placed on the appearance of the nuclear changes. It has not actually been established that the string of granules belongs to the nucleus. They may in reality be extra-nuclear structures. As regards the formation of daughter individuals by a process of budding, it must be remembered that this process is not peculiar to the piroplasmata.

It is of general occurrence amongst the Sporozoa, for when merozoites are being formed, this usually takes place by the formation of small buds on the surface of the schizont, which gradually increase in size till they are fully formed. The merozoites remain attached to the remains of the cytoplasm of the parent for some time, till finally it is completely absorbed into the merozoites or the merozoites separate from a residual body. Sometimes the buds are formed from the ends of the schizont only, while at other times they arise uniformly all over the surface. The process occurs in the coccidia, and is seen also in the malarial parasites. The formation of the sporozoites in the oöcysts of malarial parasites takes place in a similar manner, so that it is exceedingly doubtful if there ever occurs a sudden segmentation into merozoites or sporozoites, as descriptions often imply. The process, as seen in *B. bigemina*, is peculiar in that only two, instead of the usual large number of buds, are formed.

The number of parasites present in the blood of an animal infected with *B. bigemina* varies considerably. There may be very few, so that they are found with difficulty, or they may occur in over 50 per cent. of the red cells. It is only occasionally that as many as four parasites occur in a single cell. As is the case with other species, *B. bigemina* remains in the blood of clinically recovered animals for many years after its apparent disappearance, as tested by microscopical examination, for inoculation of blood will bring about infection in animals which have not previously had the disease.

FIG. 409.—*Margaropus annulatus* (♀), THE TRANSMITTER OF *Babesia bigemina* AND *B. mutans* (× 10). (ORIGINAL.)

Transmission. — The transmitting hosts of *B. bigemina* are ticks of the genus *Margaropus* (*Boophilus*). Smith and Kilborne (1893) in America demonstrated the rôle of *M. annulatus* (Fig. 409). This tick is limited to the districts south of latitude 35°, which is also the northern limit of hæmoglobinuric fever of cattle. In South Europe and the Caucasus, *M. calcaratus*, which is confined to areas south of latitude 44°, appears to be the vector. In Central Africa, *M. decoloratus* was proved to convey the disease by Koch (1898, 1898a), and Laveran and Vallée (1905). In North Africa, *M. calcaratus* was found to be the vector by Brumpt (1920), while *M. australis* was incriminated in Australia by Pound and Hunt (1895), in Venezuela by Lignières (1901) and Ziemann (1902), and in Brazil by Brumpt (1920). With ticks belonging to genera other

than *Margaropus*, some positive transmission experiments have been recorded. Theiler (1909) succeeded with *Rhipicephalus appendiculatus*, a tick which, unlike the species *Margaropus*, lives on different hosts in the larval, nymphal, and adult stages of its development. Larvæ or nymphs taken off infected cattle produced infections in other cattle on which they were placed after becoming nymphs or adults respectively. This tick also appears to be a vector in South and Central America.

Clark (1918) found that in Panama the white-tailed deer (*Odocoileus chiriquensis*) occasionally harbours *B. bigemina*, as also do cattle in this locality. Clark and Zetek (1925) collected large numbers of ticks (*M. australis*) from slaughtered cattle, in the brain smears of which *B. bigemina* was demonstrated. The offspring of eggs laid by these ticks were then allowed to feed on a healthy calf and on a deer. Both animals acquired a mild infection of *B. bigemina*. It appears, therefore, that the tick is responsible for spread of infection amongst cattle and deer.

In Northern Europe, Knuth (1915) has noted that a parasite of cattle morphologically indistinguishable from *B. bigemina* is transmitted by *Hæmaphysalis punctata*. Ticks of the genus *Margaropus*, the usual vectors of *B. bigemina*, do not extend so far north, and this fact, together with the frequency of death of infected animals from rupture of the spleen, has led to the view that the organism is a distinct species. Nicolle and Adil Bey (1899) noted that in Constantinople cattle infected with *B. bigemina* not infrequently died from the same accident, so that there is no real evidence that a distinct species is represented, as indeed Brumpt (1920) points out.

Cycle in the Tick.—Very little is known of the actual development in the tick. Koch (1906a) noted that in the stomach of the tick the pear-shaped forms escaped from the red cell, became amœboid, and threw out long spiky pseudopodia (Fig. 410, A-J). These amœboid forms were described as associating in pairs, giving rise to elongate bodies having two nuclei, and spiky pseudopodia at their extremities. Fusion of the two nuclei was then supposed to take place, and the pseudopodia were withdrawn. Forms of a similar nature were described by Dschunkowsky and Luhs (1909, 1909a), but it seems very doubtful if the amœboid forms are essential stages in the development, and in his account of the development of *B. canis* in the tick Christophers expresses the same opinion.

Symptoms and Pathology.— The disease, red-water fever, occurs in an acute or chronic form. Cattle first show symptoms from one to two weeks after exposure to infection. There is fever and passage of urine coloured red by hæmoglobin, resulting from the rapid destruction of red blood-corpuscles and the excretion of hæmoglobin by the kidneys. The blood becomes thin, pale, and watery, and the number of red cells falls from a normal of seven million per cubic millimetre to one million or even

less. The animals quickly become jaundiced, owing to the conversion of excess of free hæmoglobin into bile by the liver. Death may occur in a week or ten days, or a gradual recovery may take place. At *post-mortem*
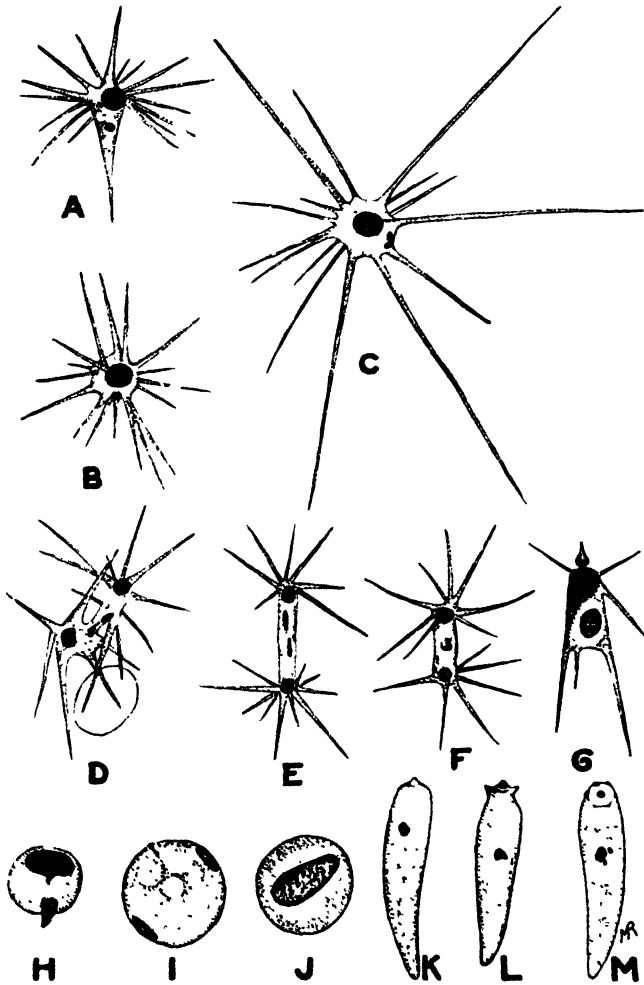
FIG. 410.—DEVELOPMENTAL STAGES OF *Babesia bigemina* AND *Babesia canis* IN TICKS, AS DESCRIBED BY KOCH AND CHRISTOPHERS (× 2,000). (A-J AFTER KOCH, 1906; K-M AFTER CHRISTOPHERS, 1907; FROM MINCHIN, 1912.)

A-C. Forms of *B. bigemina* with radiating pseudopodia.　　　　D-G. Union of gametes.
H-J. Spherical forms (zygotes ?).　　　　K-L. Motile zygotes (oökinetes) of *B. canis*.

the changes associated with extreme anæmia and jaundice resulting from rapid blood destruction are found. The subcutaneous tissues are stained with bile and the muscles pale. The spleen is much enlarged, and of a slaty colour externally and bluish-red in section. The liver, which is also

enlarged, is of a brownish-red or yellow-brown colour in section. The kidneys are swollen, and are reddish-brown in colour. The epithelial cells of the tubules are swollen and granular, while the lumens of the tubules contain casts and granules of bood-pigment. The bones show extension of the red marrow, which is deeply congested.

If animals survive the acute stage of an infection with *B. bigemina* they recover, after which it is often impossible to discover parasites by direct examination, though by inoculation of blood to new animals the persistence of parasites for many years can be demonstrated. As was first shown by Nicolle and Adil Bey (1899), any intercurrent infection is liable to light up these latent infections, so that parasites again become
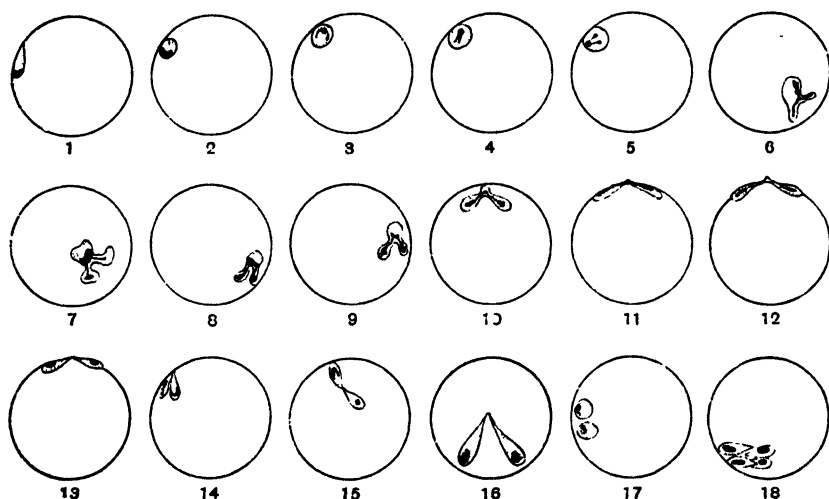
FIG. 411.—*Babesia bovis* OF CATTLE: METHOD OF MULTIPLICATION BY BUDDING IN RED BLOOD-CORPUSCLES (×*ca.* 3,000). (AFTER NUTTALL, 1913, FROM *Parasitology*, vol. vi., p. 305.)

plentiful in the blood. This fact has to be borne in mind when the organisms are discovered in the blood of sick animals, as their presence is not necessarily an indication of a recent infection.

Babesia bovis Starcovici, 1893.—Babes (1888) described the organisms which he discovered in the blood of cattle which had died of hæmoglobinuric fever. According to Vrijburg (1918) and Brumpt (1920), Babes was dealing with mixed infections of two distinct types of piroplasma, *B. bovis* and *B. bigemina*, for one of which Starcovici's specific name may be retained. *B. bovis* is a parasite of European cattle, and occurs in all parts of the Continent.

Morphology.—*B. bovis* is a comparatively small organism, the largest forms having a diameter of not more than 1·5 microns (Plate XVIII.,

11-15, p. 986, and Fig. 411). The nucleus, as seen in dried films stained by Romanowsky stain, appears as a red dot, extending from which may be a string of fine granules. There occur round, ovoid, pear-shaped, or more irregular parasites, and there is a tendency for them to be situated near the margin of the red cells. Two pear-shaped individuals are often seen lying with their pointed ends near together or in contact, and in this condition the blunt ends diverge considerably from one another or the two parasites are in line, the blunt ends pointing in opposite directions. It was on this account that M'Fadyean and Stockman (1911) proposed the name *Piroplasma divergens* for this parasite. Very small forms also occur in which very little cytoplasm can be detected, and Brumpt states that in some nothing but the chromatin granule is visible, the parasite then having the form of an ana-

plasma. *B. bovis* is a distinctly smaller parasite than *B. bigemina*, which it resembles in the method of its multiplication. Sohns (1918) suggested the name *Microbabesia divergens* for this parasite, while Mesnil (1919) suggested for it and other similar forms of inter-mediate size the generic name *Babesiella*.

Stockman and Wragg (1914) were able to demonstrate that animals which had recovered from infections due to *B. bigemina* were still liable to infection with *B. bovis*

FIG. 412.—*Ixodes ricinus* (♀), THE TRANS-MITTER OF *Babesia bovis* (× 20). (ORI-GINAL.)

(*P. divergens*), and Brumpt has shown that animals which have recovered from an infection of *B. bovis* are still susceptible to *B. argentina*.

Transmission.—In nature *B. bovis* is carried by the tick, *Ixodes ricinus* (Fig. 412), as was first shown by Kossel, Weber, Schütz and Miessner (1904). These observers infected cows by exposing them to the bites of larvæ which had developed from eggs laid by ticks collected from infected animals. They also showed that if larvæ were allowed to feed on infected animals, the resulting nymphs were capable of transmitting the infection, though, as Brumpt remarks, the larvæ may have been already infective before feeding. Brumpt was able to produce an infection in a calf by allowing it to be bitten by a nymph which was either hereditarily infected or had contracted an infection as a larva. Stockman (1908), using the tick *Hæmaphysalis punctata* in England, was able to transmit an infection by means of adults which had fed as nymphs on infected animals, but

there is some doubt as to whether he was working with *B. bovis* or *B. bige-mina*. It seems clear, therefore, that *Ixodes ricinus* is the normal trans-mitting host, and that the virus passes through the egg of the adult tick to the succeeding generation. Nothing is known of the development in the tick.

Symptoms and Pathology.—Febrile symptoms appear in five to twenty-eight days after exposure to infection, and two to three days later hæmoglobinuria occurs. This is followed by progressive anæmia, accom-panied by general weakness. The parasites occur in large numbers in the blood. The symptoms are much milder in young than in old animals, and hæmoglobinuria is seldom observed in those under eighteen months old. The mortality varies from 6 to as much as 50 or 60 per cent., according to the locality and the conditions prevailing. *Post-mortem* examinations reveal congestion of the organs and marked enlargement of the spleen and liver. The kidneys are deep red in colour, and on section show degenera-tion of the epithelium of the tubules, which are blocked with cells, casts, and débris containing granules of hæmoglobin. The pyloric opening of the stomach and the mucous lining of the intestine show numerous ecchymoses.

Parasites which may be merely Races of B. bovis. — Lignières (1901) in the Argentine gave the name *B. argentina* to a parasite which closely resembles *B. bovis*. According to Brumpt (1920), it differs in that the organisms are always scanty in the blood, and may be difficult to find, while they do not show the same tendency to be near the margin of the red cell. The same types of parasite occur in the two species, which could not be definitely distinguished were it not for the evidence of immunity. Vrijburg (1918), while studying *B. bovis* in Holland, came to the conclusion that it was identical with *B. argentina*, and that the immunity test was an insufficient basis for the separation of the species. Sohns (1918), in the Dutch East Indies, stated that *B. bovis*, *B. divergens*, and *B. argentina* were one species, and, ignoring the rules of nomenclature, proposed to rename the parasite *Microbabesia divergens*.

Lignières (1901) proved that *B. argentina* was conveyed by the tick, *Margaropus australis*. Working in France, Brumpt (1920) was able to infect animals with ticks of this species which he had received from Brazil. The symptoms are similar to those produced by *B. bovis*, but the mortality rate appears to be higher. Anæmia is never intense, even in cases in which hæmoglobinuria is very pronounced. Brumpt showed that animals which had recovered from an infection of *B. bovis* were susceptible to infection with *B. argentina*. Lignières stated that infections with *B argen-tina* and *B. bigemina* protected against each other.

Sergent, Ed., and his collaborators (1924), working in Algeria, have noted a piroplasmosis of cattle which is due to a parasite resembling

B. bovis in size, but differing from it in that the parasites occur in the blood only in small numbers, even during the acute stage of an infection; that the forms at the margins of the red cells, so common in *B. bovis* infections, are absent; and that trypan blue fails to have any therapeutic action. On this account they regard it as a new species, which they name *Babesiella berbera*, using the name *Babesiella bovis*, as suggested by Mesnil (1919), for the well-known form, *Babesia bovis*, described above. *Babesia berbera* differs from *B. argentina* in that the symptoms produced by experimental inoculation are mild, that anæmia follows an acute attack, that trypan blue has no action, and that an infection does not protect against *B. bigemina.*
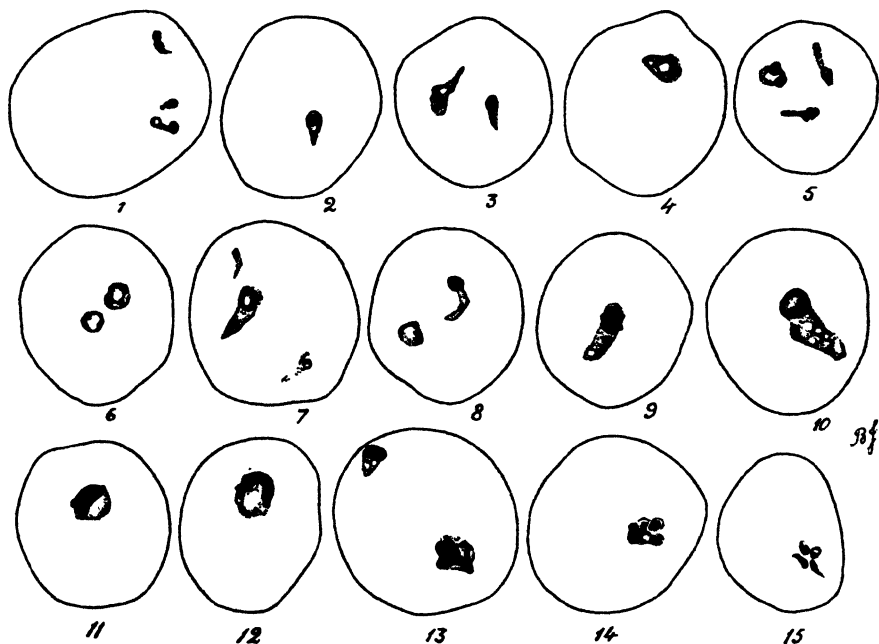
FIG. 413.—*Babesia mutans* FROM THE BLOOD OF CATTLE (× *ca.* 4,000). (AFTER GONDER, 1911.)

These features are, however, physiological ones, which are of very doubtful value for the separation of true species.

Babesia mutans (Theiler, 1906).—This is a very small parasite which is frequently found in cattle in association with *B. bigemina*, a fact which gave rise to considerable confusion till it was recognized as a distinct species by Theiler (1906*a*), who named it *Piroplasma mutans*. On account of its minute size, it is a difficult organism to study. It occurs in a multiplicity of forms, but these may be grouped as round, ovoid, comma-shaped, bacilliform, coccal, dumb-bell-shaped, ring, or cross forms (Plate XVIII., 16-20, p. 986, and Fig. 413). Each parasite, as seen in

Romanowsky stained films, consists of a blue staining cytoplasm and a chromatin area, but the latter in the smallest forms often occupies the bulk of the parasite, and in some an anaplasma-like form results, in which the cytoplasm can hardly be distinguished. The largest forms are barely 1 micron in diameter, so that there is considerable difficulty in making out the exact structure and method of multiplication. It appears that reproduction is effected by a division into two, of which the dumb-bell form is a stage, or by a division into four resulting in the cross forms, in which four minute pear-shaped individuals radiate from a central point. That reproduction takes place in the peripheral blood is proved by the result of inoculation experiments. In the case of other piroplasmata, and, indeed, of all the blood parasites which reproduce in the peripheral blood, inoculation of blood containing these reproducing forms conveys infection to healthy animals. This occurs in the case of *B. mutans*. As will be shown below, *Theileria parva*, the blood forms of which closely resemble those of *B. mutans*, can only be inoculated by means of the blood when it contains schizonts, which are usually confined to the internal organs.

As regards its effect on animals, *B. mutans* is a benign parasite. It produces few, if any, symptoms, and never gives rise to hæmoglobinuria, though it may be the cause of a certain amount of anæmia, but even this is doubtful. The parasitized red cells are not visibly altered by its presence, and, as a rule, only a small percentage of the cells is infected. *B. mutans* is with difficulty distinguished from *T. parva* by blood-film examinations alone. As pointed out by Sergent and his co-workers (1924), one attack does not confer immunity, while it is possible to produce a superimposed infection.

The infections due to *B. mutans* are generally less intense than those due to *T. parva*. The schizonts which occur in the spleen, bone marrow, and lymphatic glands in cases of *T. parva* infections do not occur in the case of *B. mutans*, though this statement will require qualification if the observations of Brumpt, Velu, and Doyle (p. 1035) are confirmed. They believe that the parasite is a *Theileria*, as blue bodies occur in the organs in small numbers in intense infections.

B. mutans is widely distributed through South Europe, Asia, Africa, and Australia. According to Brumpt, it has not been found in America. Theiler (1907), who first recognized *B. mutans* as a distinct species, was unable to transmit infection through the agency of ticks of the genus *Margaropus*, which are the usual vectors of *B. bigemina*. Later (1909), he was successful with *Rhipicephalus evertsi* and *R. appendiculatus* (Fig. 429). With these ticks, however, there is no passage of the virus through the egg. It is taken up in the nymphal stage and transmitted at the succeeding stages of development when a new host is attacked.

Other Species of Babesia in Cattle.

Other species of *Babesia* have been described from cattle, but it is probable that these are merely varieties of the well-known types. The forms named *Babesia argentina* and *B. berbera* have been mentioned above. Bowhill (1909), who had studied *B. bigemina* in Africa, described as *Piroplasma hudsonius bovis* a form seen in cattle in British Columbia. The disease produced was of a relapsing nature, and proved fatal after several relapses. The parasites were scanty in the blood, and the typical pairs of pear-shaped individuals, so characteristic of *B. bigemina*, were very few in number. Bowhill associated ticks of the genus *Dermacentor* with this disease, and Brumpt concludes that the species must be *D. venustus*, which is a common tick of North American cattle.

Macfie sent a blood-film of a West African cow to França for inspection. It contained a piroplasm, which Macfie considered to be peculiar. França (1918*a*) noted the presence of *B. bigemina* and *B. mutans* in these films, and in addition other forms which did not appear to belong to either of these. There were elongate individuals stretching across the cell, very irregular amœboid forms, and rounded and pear-shaped individuals, all of which were larger than the usual forms of *B. bigemina*. The nucleus of the parasite consisted of a single compact chromatin body, and thus differed from the nucleus of the typical forms of *B. bigemina*, which, in addition to the compact chromatin body, has a string or group of finer granules associated with it. Since two well-known species were present in the blood at the same time, the possibility of the unusual forms being merely peculiar types of these has to be considered. França, however, regarded them as belonging to a different parasite, which he placed in the genus *Achromaticus* under the name *A. macfiei*. It is evident that further investigation is required before the validity of the species can be accepted. Schein (1921) described as *P. bubali* a parasite of Annam buffaloes. This form closely resembles *B. bigemina*, and may be identical with it, though Schein (1923) claims that it is distinct owing to his failure to inoculate typical *B. bigemina* of a calf into a buffalo. Schein also describes a small parasite of the buffalo. This, he says, differs in certain respects from *B. mutans*, but resembles the *Nuttallia (B. equi)* of horses. Neveu-Lemaire (1912) gave the name *P. buffeli* to a small parasite of the buffalo.

The piroplasma, which was described by Carpano (1915) as occurring in cattle of North Tripoli, and *P. annulatum*, which Dschunkowsky and Luhs (1904, 1909) recorded from Transcaucasia, probably represent mixed infections of *Theileria parva*, *B. mutans*, and *Anaplasma*. Mason (1922), however, believes that Egyptian fever of cattle may be the same as the disease studied by Dschunkowsky and Luhs, and as the parasite

producing it belongs to the genus *Theileria*, but differs in certain respects from that of East Coast fever, he regards it as *T. annulata*, which is discussed more fully below (p. 1034).

BABESIA OF SHEEP AND GOATS.

As pointed out above, Babes (1892) noted that sheep suffering from a disease known as "carceag" in Roumania harboured a parasite similar to the one described by him previously as the cause of "red water" of cattle. This organism was named *Babesia ovis* by Starcovici (1893). Since that time it has become clear that sheep and goats are liable to infection with three species of *Babesia* corresponding with *B. bigemina*, *B. bovis*, and *B. mutans* of cattle, in addition to a species of *Theileria*. It might be conjectured that the parasites of sheep and goats are identical with those of cattle, but inoculation experiments have so far failed to produce cross-infection. It is impossible from Babes's description to identify with certainty the form observed by him, but, as in the case of the parasites of cattle, he may have been dealing with mixed infections.

Laveran and Nicolle (1899*x*), employing the name *Piroplasma ovis*, described a parasite of sheep in Constantinople. Their description shows that the parasite was smaller than *B. bigemina* of cattle previously studied by them (1899). They definitely state that the parasite of sheep never exceeds 2 microns in diameter, so that it is highly probable that they were dealing with the organism of intermediate size, the name for which can be accepted as *B. ovis* (Fig. 414, 5-8). Dschunkowsky and Luhs (1909) gave the name *P. hirci* to a parasite of goats. Du Toit (1918) assumes that this parasite corresponds with *B. mutans* of cattle, and places it in his genus *Gonderia*, but from the figures given by Dschunkowsky and Luhs, and their statement that the animals suffered from hæmoglobinuria, this conclusion is not justified. It seems clear that *P. hirci* is the parasite of intermediate size in the goat, and the name becomes a synonym of *B. ovis*. Ratz (1913), working in Hungary, described under the name *B. ovis* both the small parasite and the one of intermediate size. Lestoquard (1924), using the generic name *Babesiella* suggested by Mesnil (1919), named the form of intermediate size *Babesiella ovis*.

As regards the parasite corresponding with *Babesia mutans* of cattle, it was seen by a number of observers, most of whom, without adequate evidence, referred to it as *Theileria ovis*. On the assumption that it belongs to the genus *Babesia*, there is no available specific name for the parasite, which was first properly described by Sergent, Parrot and Hilbert (1922), who named it *Gonderia ovis*. It may appropriately be named *Babesia sergenti* (Fig. 414, 1-4).

The large parasite of sheep was first clearly observed by Motas (1903), who stated that the form he studied was morphologically identical with *B. bigemina* of cattle. It was later described and figured by Dschunkowsky and Luhs (1909), and by Lestoquard (1925), who referred to it as *Piroplasma ovis*. As the piroplasmata of sheep all belong to the genus *Babesia*, there is no name available for the large form, which may conveniently be named *Babesia motasi* (Fig. 414, 9-12).

Owing to the fact that the various parasites of sheep have been only recently differentiated, many of the earlier records contain no indication
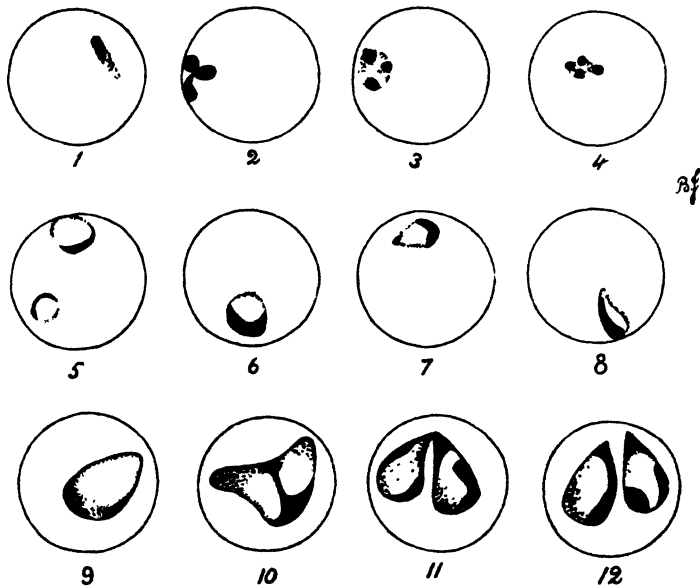
FIG. 414.—THREE SPECIES OF *Babesia* IN BLOOD OF SHEEP (× 3,000). (AFTER LESTOQUARD, 1924 AND 1925.)

1-4. *Babesia sergenti.*   5-8. *Babesia ovis.*   9-12. *Babesia motasi.*

of the particular organism observed. Piroplasmosis of sheep was discovered by Babes in Roumania. It was observed by Laveran and Nicolle (1899) in Turkey, by Bonome (1895) in Italy, by Motas (1903, 1903a) in Roumania, by Dschunkowsky and Luhs (1909) in Transcaucasia, by Inchiostri (1912) in Dalmatia, and by Markoff (1916) in Bulgaria. Ziemann (1902) stated that he had seen a piroplasm in films made from the blood of Venezuelan sheep, while Yakimoff (1917a) and Yakimoff and Schokhor (1917) record large parasites from sheep and goats in Turkestan. The records by Johnson (1903) in America, and by Hutcheon and Robertson (1902) in South Africa, are, according to Nuttall (1913), open to question.

Lestoquard (1924, 1924a, 1925) has studied the infection in Algeria, and clearly differentiated the various forms which he has proved to be infective to both sheep and goats.

Babesia motasi n. sp.—As noted above, this parasite was first definitely seen in sheep by Motas (1903). Dschunkowsky and Luhs (1909) clearly figured this form, which has recently been described by Lestoquard (1925) in Algeria (Fig. 414, 9-12). The last-named observer finds that it occurs also in goats, and can readily be inoculated from animal to animal.

The disease produced by *B. motasi*, like red-water fever of cattle, may occur in an acute or chronic form. The acute disease, according to Motas, is accompanied by fever, prostration, hæmoglobinuria, and profound anæmia. The number of red blood-corpuscles may fall rapidly from eight to one and a half millions. Death often results. The chronic form of the disease may pass unrecognized unless the parasites are discovered by blood examination.

Morphologically *B. motasi*, according to Lestoquard (1925), corresponds very closely with *B. bigemina* of cattle. The pear-shaped parasites are the forms most frequently seen. They are single or arranged in pairs. Ovoid, round, or irregularly shaped parasites also occur. They measure from 2·5 to 4 microns in length by 1·2 to 3 microns in breadth. As in the case of *B. bigemina*, when two pear-shaped forms are united by their pointed extremities, the angle between them is an acute one (Fig. 408). In the case of *B. ovis*, in which pear-shaped forms are rarely seen, the angle between the individuals of a pair is obtuse, as in *B. bovis* (Fig. 411). The infections produced by *B. motasi* are heavier than those due to *B. ovis*. The nucleus, or rather the chromatin, often appears double in *B. motasi*, while it is invariably single in *B. ovis*. In the goat, *B. motasi* produces a hypertrophy of the red blood-corpuscles.

Animals which have been infected with *B. motasi* and have recovered from the acute disease are readily infected with *B. ovis*. Trypan blue has a definite action on infections due to *B. motasi*, but not on those due to *B. ovis*.

Transmission of *B. motasi* is brought about by the tick, *Rhipicephalus bursa*, as clearly demonstrated by Motas (1903, 1903a). Though the tick is a one-host tick, it appears that actual transmission is not effected till the infected young which hatch from the eggs have reached the adult stage of their development. Dschunkowsky and Luhs (1909) reported that in the intestine of the tick the parasites assumed an amœboid form with spiky pseudopodia, like the developmental stages of *B. bigemina* described by Koch (Fig. 410).

Babesia ovis Starcovici, 1893.—It seems probable that this parasite was first observed by Babes (1892), though Laveran and Nicolle (1899) gave the first recognizable description (Fig. 414, 5-8). The parasite of goats,

named *Piroplasm hirci* by Dschunkowsky and Luhs (1909), is undoubtedly *B. ovis*, and is not allied to *B. mutans*, as du Toit (1918) conjectures. It was definitely figured by Ratz (1913), and has been studied by Lestoquard (1924) in Algeria.

The chief differences between *B. ovis* (called *Babesiella ovis* by Lestoquard) and *B. motasi* have been noted above. It is smaller than *B. motasi*, the largest diameter varying from 1 to 2·5 microns in length. Pear-shaped individuals are not frequently encountered. When arranged in pairs, the angle between them is obtuse, as in *B. bovis*, while they show a tendency to occupy the margins of the red blood-corpuscles (Fig. 411). The majority of the parasites are round. As a rule, not more than 0·6 per cent. of the red blood-corpuscles are infected.

The disease produced by *B. ovis* is less severe than that due to *B. motasi*. The acute phase is accompanied by fever, jaundice, and progressive anæmia.

No precise information regarding the ticks involved in the transmission of *B. ovis* is available.

Babesia sergenti n. sp.—The small parasite of sheep (Fig. 414, 1-4) was definitely figured by Ratz (1913). He referred it to *Piroplasma ovis*. It was studied in Algeria by Sergent, Parrot and Hilbert (1922), and by Lestoquard (1924), who adopted the name *Gonderia ovis*. As a result of the work of the last-named observer, it appears clear that the small parasite of goats is identical with that of sheep, so that the name *P. hirci*, given to the goat parasite by Dschunkowsky and Luhs (1909), would have priority if du Toit's conclusion that they were dealing with a parasite of the *B. mutans* type were correct. It is clear, however, from the figures, which show definite pear-shaped forms of the correct size arranged in couples, and from the description of the disease produced, that they were dealing with a parasite of the *B. bovis* type. Yakimoff (1916) states that he called attention to this parasite in Transcaucasia in 1913, and designated it *Theileria ovis*. Schellhase (1913, 1914) observed it in sheep and goats in West and East Africa, and Macfie (1914a) in sheep in the Gold Coast, while the writer (1915d) saw it in blood-films from sheep in Rhodesia. Rodhain (1916) recorded it in the Belgian Congo, and referred to it as *T. ovis*, while Yakimoff and Paroïsky (1917) observed it in Turkestan. In none of these cases was there any evidence that the parasite belonged to the genus *Theileria*.

As in the case of *B. mutans* of cattle, *B. sergenti* produces no recognizable symptoms in sheep and goats. Lestoquard (1924) in Algeria found, by examination of single films, 130 sheep infected out of 202 examined. The infections are never intense, only 0·1 to 0·3 per cent. of the red blood-corpuscles being infected.

Morphologically *B. sergenti* corresponds very closely with *B. mutans*. Similar rounded and bacillary forms occur. Reproduction is by budding into two or four, so that the characteristic cross forms are produced. Nothing is known of the method of transmission.

BABESIA OF PIGS.

Sparapani (1917) recorded piroplasmata of the *Piroplasma ovis* (? *B. motasi*) type in three pigs which had died in Italy. As the pigs three months before had fed upon the flesh of sheep which had died of piroplasmosis, he supposed they had acquired the infection at that time. Knuth and du Toit (1921) state that Trautmann in 1914 observed piroplasmosis of pigs in East Africa. They reproduce some of the figures from his still unpublished report, and these show parasites resembling *B. bigemina* or *B. bovis*. They give the name *P. trautmanni* as a new species, but the name had already been given by du Toit (1918) to what was evidently Trautmann's parasite.

BABESIA OF HORSES AND OTHER EQUIDÆ.

Piroplasmosis of horses appears to have been first recognized by Dupuy in 1888 in Senegambia, but Guglielmi (1899) was the first to see parasites in the blood. The organism was recognized as a piroplasma, and named *P. equi* by Laveran (1901). Nuttall and Strickland (1910) showed that two distinct parasites occurred in horses. One was a small form which was originally seen by Laveran, and which they placed in a new genus as *Nuttallia equi*, and the other a larger form of the *B. bigemina* type, which they referred to as *Piroplasma caballi* Nuttall. Both these forms are here retained in the genus *Babesia*.

Babesia caballi (Nuttall, 1910).—This form, like others of its type, produces hæmoglobinuric fever of horses (Plate XVIII., 21-25, p. 986, and Fig. 415). It occurs in South Europe, and extends through the Caucasus into Asia. It has been noted by Marzinowsky and Bielitzer (1909) in Russia; by Dschunkowsky and Luhs (1913), and Yakimoff, Schokhor and Koselkine (1917) in Turkestan; by Carpano (1913b) in Italy; by Darling in Panama (1913); by Valladares (1914) in Madras; and by Velu (1918) in Morocco.

The parasites are relatively large, and resemble *B. bigemina* of cattle (Plate XVIII., 21-25, p. 986, and Fig. 415). Multiplication takes place by the budding process which has been described above, and pairs of pear-shaped forms are to be found in the cells.

Marzinowsky and Bielitzer (1909), working in South Russia, proved that transmission was effected by the tick, *Dermacentor reticulatus*, while Carpano (1913b) in Italy claims that *Margaropus annulatus* is the vector.

Babesia equi (Laveran, 1901).—This is the form which was first seen in horses, and it is much more widely distributed than *B. caballi* (Plate XVIII., 26-30, p. 986, and Fig. 416). It produces a disease like that caused by *B. caballi*, and occurs in Africa, South America, Southern Europe, Transcaucasia, and parts of Southern Asia. According to Carpano, who studied both forms in Italy, *B. equi* produces a more serious disease than *B. caballi*. It has been recorded by Dschunkowsky and Luhs (1913) from Transcaucasia, by Carpano (1913b) in Italy, by Valladares (1914) in Madras, by Schein (1917) in Annam, and Yakimoff, Schokhor and Koselkind (1917) in Turkestan. Both this form and *B. caballi* were seen by the writer in Macedonia in 1918.
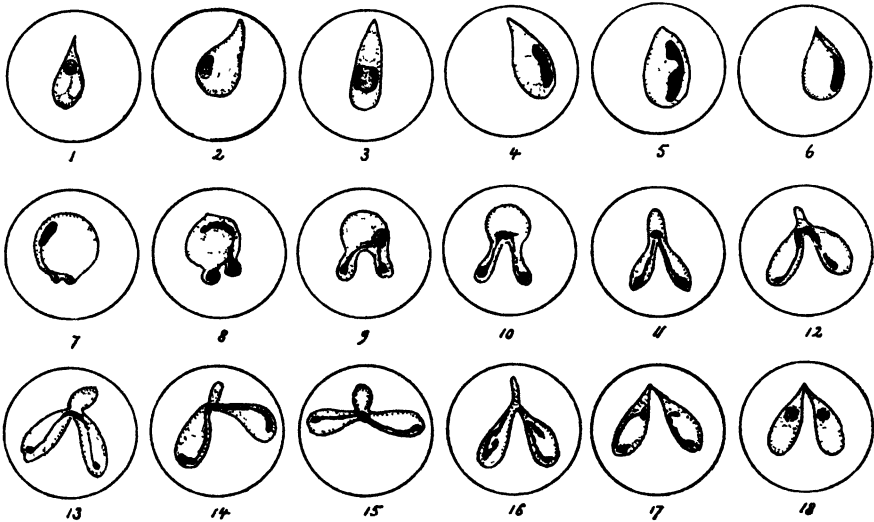
FIG. 415.—*Babesia caballi* OF THE HORSE: METHOD OF MULTIPLICATION IN RED BLOOD-CORPUSCLE BY BUDDING PROCESS (× *ca.* 3,000). (AFTER NUTTALL AND STRICKLAND, 1912; FROM *Parasitology*, vol. v., p. 78.)

Nuttall and Strickland (1910) and du Toit (1919) showed that horses which had recovered from infections due to *B. caballi* were liable to infection with *B. equi*. On account of the small size of this parasite, and the fact that when division takes place four daughter individuals are formed, and that these may remain attached to one another in the form of a cross, França (1909) established a new genus (*Nuttallia*) for its reception. As already remarked, the difference in size and in the number of daughter individuals produced are not sufficient grounds for the recognition of a new genus. *B. equi* is a small parasite, the largest forms being barely 2 microns in diameter (Plate XVIII., 26-30, p. 986, and Fig. 416). According to Nuttall and Strickland (1910), the small slightly elongate individuals resulting from division escape from the cell and enter other cells. Here growth into pear-

shaped forms about 2 microns in length takes place. These become irregular in shape, and finally rounded, whereupon division commences. The nucleus divides into two parts, and then each of these divides again. Finally, the cytoplasm buds off four daughter forms, which radiate

Cycle

showing the usual mode
of multiplication
of

Nuttallia equi

in the circulating blood
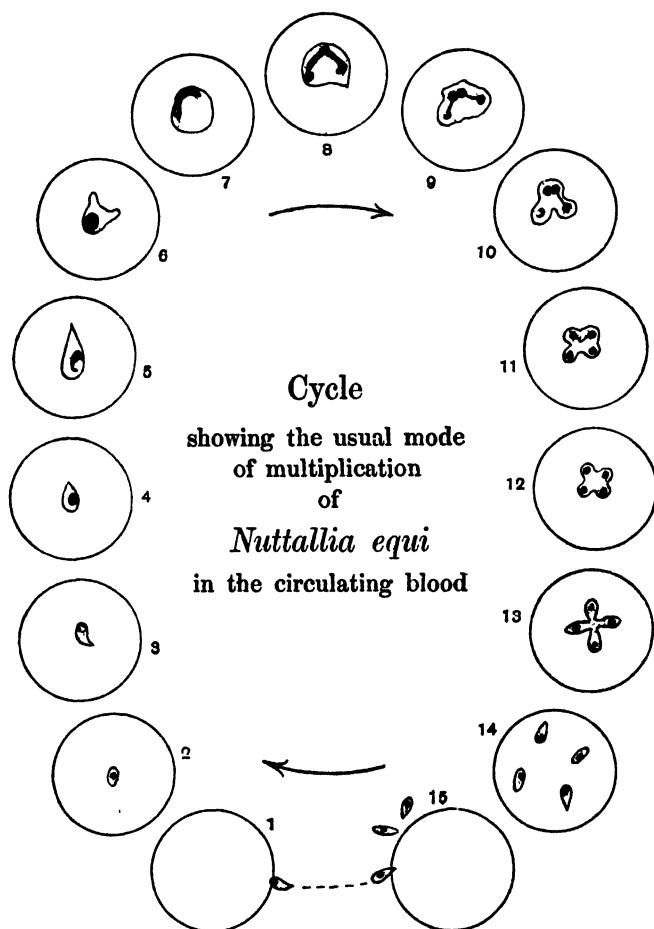
FIG. 416.--*Babesia equi* (*Nuttallia equi*) FROM THE HORSE: CYCLE OF DEVELOPMENT IN THE BLOOD (× *ca*. 3,000). (AFTER NUTTALL AND STRICKLAND, 1912, FROM *Parasitology*, vol. v., p. 75.)

1. Invasion of red blood-corpuscle by form free in plasma.   2-5. Growth of parasite.
6. Large, actively amœboid form.   7-10. Division of nucleus.
11-15. Budding process and escape of four daughter forms from corpuscle.

from a central point, giving rise to the characteristic cross forms. The four daughter forms eventually separate and escape from the red cell to infect other cells.

The South African form of equine piroplasmosis, which is due to *B. equi*, was shown by Theiler (1905a) to be transmitted by *Rhipicephalus*

evertsi (Fig. 429). This observer showed that infections could also be produced in mules and donkeys. Young animals, though acquiring the disease, are not so seriously affected as older ones. After clinical recovery, the blood remains infective for many years. Animals which have recovered are known as " salted animals," and the blood of salted young horses is inoculated into young animals as a protective measure. A mild type of the disease is produced, after which the animals are not susceptible to re-infection. Carpano (1913*b*) states that in Italy *B. equi* is transmitted by *Rhipicephalus bursa*.

Possibility of Other Species of Babesia in Equidæ.—As was noted by Theiler in South Africa, a parasite similar to *B. equi* occurs in the mule and donkey. He also (1907*a*) noted it in a zebra. Dale (1903) reported it in donkeys in the Transvaal. Bouet and Roubaud (1912*b*) recorded such a form in donkeys of West Africa, Dschunkowsky and Luhs (1913) in donkeys and mules in Transcaucasia, Carpano (1914) in these animals in Italy, Schellhase (1914) and Griffiths (1918) in donkeys in East Africa, and Yakimoff (1917*a*) in donkeys in Turkestan. The parasite of the donkey was named *Nuttallia asini* by Dschunkowsky and Luhs (1913). A form seen by Yakimoff (1917*a*) in camels of Turkestan, and named by him *Theileria camelensis*, is possibly the same as *B. equi*, which was seen by Yakimoff, Schokhor and Koselkine (1917) in horses and donkeys in the same district. There is no evidence that it belongs to the genus *Theileria*. Lingard and Jennings (1904) had reported these parasites from camels in India. Manleitner (1912) saw similar forms in two captured giraffes in East Africa. The animals appeared to have died of piroplasmosis.

BABESIA OF DOGS.

In the year 1895 Piana and Galli-Valerio showed that a form of jaundice which attacked hunting dogs in Lombardy was due to the presence of a piroplasma in the blood. Celli (1900) in Italy observed the same parasite in dogs imported from Lombardy, after which it was reported from France by Leblanc (1900), Almy (1901), Nocard and Almy (1901), and Nocard and Motas (1902). Yakimoff (1910) recorded it from Russia. Hutcheon (1893) had recognized the disease in dogs in South Africa, but Robertson (1901) was the first to record the parasite in this country, though he states that it was actually discovered by Purvis. Koch (1897) discovered the organism in East Africa. It was seen by Marchoux (1900) in Senegal, by James (1905) in Assam, and by Webb (1906) and Christophers (1907) in India. Dschunkowsky and Luhs (1909) recorded it from the Caucasus, and the writer (1911*a*) observed it in Bagdad and later in Aleppo. Mathis (1909) records its presence in Tonkin, an observation which confirmed the claim

made by Eggebrecht (1908) that it occurred in Eastern Asia. It is evident, therefore, that the disease which was named biliary fever or malignant jaundice of dogs is widely spread through Southern Europe, Asia, and Africa. No record can be found of its occurrence in Australia, and the typical parasite appears to be absent in America, though it has been recorded by Martinez (1914) from Porto Rico, while Clark (1918) observed it in imported hunting dogs in Panama.

Seidelin (1912*a*) recorded the presence of a piroplasm in a dog in Yucatan, while Pestana (1910) had seen a form in dogs in Brazil. Owing to certain morphological peculiarities, the latter observer considered the parasite to be different from *B. canis* of the Eastern hemisphere, and named it *P. vitalii*. The organism was studied by Carini and Maciel (1914*a*), who came to the conclusion that it could not be included in the genus *Piroplasma* (*Babesia*), and placed it in a new genus, *Rangelia*.

Patton (1910) described as *Piroplasma gibsoni* a parasite which he found in dogs of the Madras hunt, and also in the jackal (*Canis aureus*). França places this form in the genus *Achromaticus*, which was founded by Dionisi for the unpigmented parasites of the red blood-corpuscles of bats. Another form was described by Nuttall (1912) as *Rossiella rossi* of the jackal (*C. adustus*) of British East Africa. Yakimoff and Schokhor (1917) recorded a large parasite of the *B. canis* type and a small one of the *B. mutans* type from wolves of Turkestan. A small form was also found in foxes. Plimmer (1915) observed a piroplasm in an Indian wild dog (*Cyon dukhunensis*) which had died in the Zoological Gardens in London. As regards the validity of the various species described, this can only be decided by further investigations.

Babesia canis (Piana and Galli-Valerio, 1895).—The disease known as malignant jaundice of dogs is of wide distribution in the Old World, and was shown by Piana and Galli-Valerio (1895) to be due to a piroplasm, which they named *Pyrosoma bigeminum* var. *canis*. It is a comparatively large organism, and as infections are readily maintained by inoculations from animal to animal, the parasite has been extensively studied (Fig. 417, and Plate XVIII., 1-5, p. 986). In countries where the disease is endemic, the native animals become infected soon after birth. The infections are slight, and serious symptoms rarely occur. On the other hand, imported dogs contract a serious form of the disease associated with heavy infections which are frequently fatal. Similar heavy infections are noted outside endemic areas in dogs which have been infected by inoculation of blood. In native dogs, as, for instance, those of Bagdad, the writer found the parasites in moderate numbers in puppies, and not at all, or only after long search, in older animals. The mortality amongst indigenous dogs is very low, but among imported dogs it may be extremely high.

Symptoms and Pathology.—In acute infections there is fever, progressive anæmia, jaundice, and hæmoglobinuria, and the disease frequently
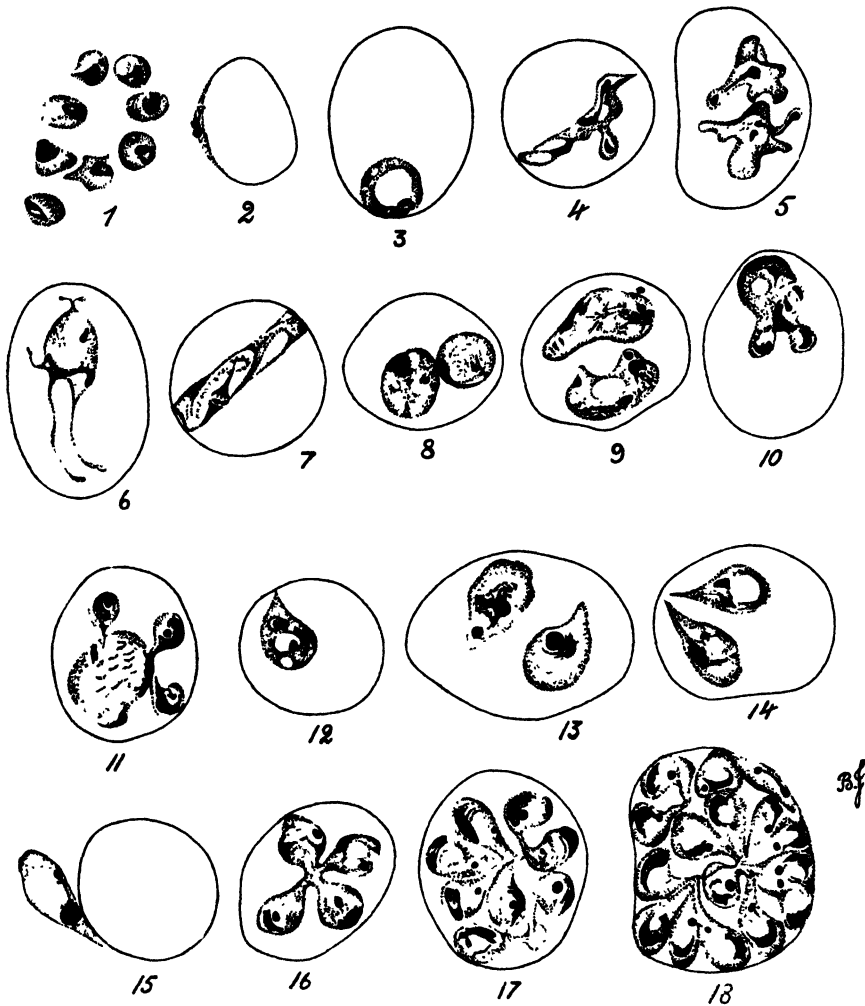
Fig. 417.—*Babesia canis* IN THE BLOOD OF THE DOG (×ca. 2,250). (AFTER KINOSHITA, 1907.)

1. Group of free forms. probably resulting from rupture of a cell with multiple infection.
2. Marginal form.   3-9. Various types of parasite.
10. Form producing two buds.   11. Form producing four buds.
12-14. Pear-shaped individuals.   15. Free pear-shaped form.
16. Form producing four buds.   17-18. Cells containing several budding parasites.

terminates fatally. In the more chronic types there may be only slight fever, a mild anæmia, and jaundice may or may not be present. Indigenous dogs, though apparently perfectly healthy, may be found to be

harbouring the parasite. Breinl and Hindle (1908) could obtain no evidence of the presence of a specific hæmolysin in the blood of infected animals. It would seem that the anæmia is due largely, if not entirely, to the destruction of red cells by the parasites. At *post-mortem* examinations, dogs which have succumbed to the disease are found to show marked jaundice of the internal organs. There is enlargement of the spleen, while the kidneys are swollen and congested. Smears of the organs, especially the lungs, liver, and kidneys, may show the parasites to be more numerous than they are in the peripheral blood. Sections of the kidneys show hyperæmia and degeneration of the epithelium of the tubules, which are filled with débris of cells, altered hæmoglobin, and granular and epithelial casts. The parasites may occur in the red blood-cells singly as round, irregular, or pear-shaped individuals. Typical budding forms leading to the characteristic pairs of pear-shaped forms are common. In heavy infections fifteen or more parasites may be found crowded together in a single cell.

Morphology. — The structure of the parasite within the red cell was studied by Nuttall (1904) and by Nuttall and Graham-Smith (1905, 1906). The typical pear-shaped individual is about 4·5 to 5 microns in length. It is pointed at one end and round and bulbous at the other. There is generally a vacuole in the cytoplasm. The nucleus, as seen in dried films stained by Romanowsky stain, consists of a deeply staining granule near the pointed end, while extending from it is a string of finer granules. Such a pear-shaped form appears to go through a definite evolution (Fig. 418). It contracts and becomes spherical, while the vacuole increases in size and the string of fine chromatin granules is withdrawn into the larger granule. Eventually the vacuole disappears, and the parasite, consisting of a cytoplasmic body and chromatin granule, passes through an amœboid phase. The commencement of division is seen in the separation of a fine granule of chromatin from the larger one, to which, however, it remains attached by a string of granules. This connection lengthens, and the smaller granule divides, as does also the connecting string of granules. While this is taking place the vacuole reappears. Eventually there is produced a rounded parasite with a granule of chromatin near its margin, while extending from it, and passing by the side of a central vacuole, is a string of granules which bifurcates to produce two limbs, which terminate near the surface of the parasite in two small granules. The cytoplasm opposite the latter now becomes elevated to form two buds, which gradually increase in size at the expense of the main mass of cytoplasm. The string of granules divides towards the large granule, till finally this divides also. The vacuole which had appeared in the earlier stage of this budding process again disappears, and a vacuole appears
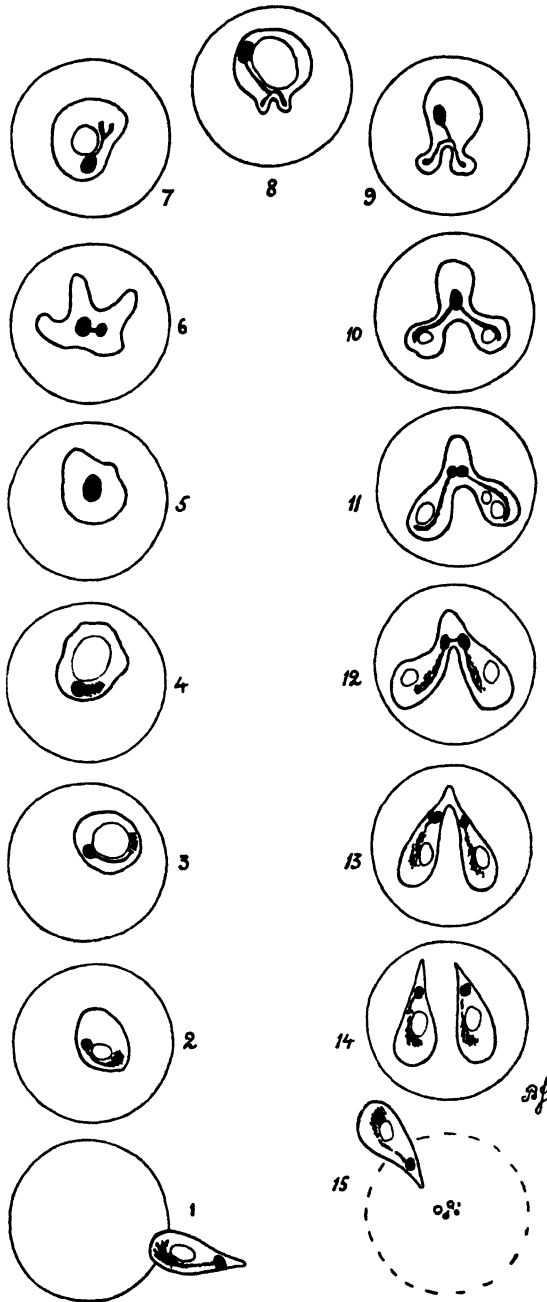
FIG. 418.—*Babesia canis* OF THE DOG: METHOD OF MULTIPLICATION BY BUDDING IN THE RED BLOOD-CORPUSCLES (× *ca.* 3,000). (AFTER NUTTALL AND GRAHAM-SMITH, 1907; SLIGHTLY MODIFIED.)

in each of the buds.   The cytoplasm of the original parasite is finally completely absorbed into the two pear-shaped buds, which remain attached to one another by their pointed extremities till final separation occurs. Each of them has the characters of the original parasite, and they may leave the cell to invade other corpuscles, or further divide to produce multiple cell infections.   Sometimes, when the two daughter individuals are only partially formed, each begins to divide so that a division into four parasites takes place.   In other cases reproduction is more irregular, and a number of buds of unequal size is formed.   The method of division described was worked out by Nuttall and Graham-Smith from observations on living parasites and stained material, and represents what may be regarded as the typical method of reproduction.   In any film of a heavy infection the parasites show a great variety of shape and size, and in many cases it may not be possible to place them in the scheme outlined above.

Schuberg and Reichenow (1912) also studied *B. canis* in the dog. They came to the conclusion that the amœboid forms are actually extra-corpuscular, since their motility is too marked for an intracellular situation. They believe that the amœboid form becomes round and produces buds, as described by Nuttall and Graham-Smith, but that the buds pass into the red cell, producing there the intracorpuscular pairs of pear-shaped parasites.   These grow at the expense of the red cell, but finally leave it to become the extracorpuscular amœboid forms.   The details of nuclear division were studied in wet fixed films (Fig. 419).   In the great majority of parasites the nucleus consists of a single rounded mass of chromatin. At division this elongates and splits into two bodies which are richer in chromatin at their ends.   A further division takes place, giving rise to two pairs of chromatin bodies, one of which in each pair is larger than the other.   A pair enters each bud, but as this increases in size the smaller of the two granules disappears, so that the nucleus of the fully-formed bud is represented by the single granule.   Though *B. canis* has been extensively studied, no one has been able to produce satisfactory evidence of the presence of gametocytes in the blood of the dog.   It has often been supposed that the pear-shaped individual is of this nature, but, as noted above, it is only a temporary phase in the life-cycle, and ultimately proceeds to division.   Christophers and Nuttall and Graham-Smith could not identify any of the blood forms as gametocytes, and as nothing is known of a sexual process in the tick, it is impossible to state if such forms are present or not.

Attempts at the discovery of a flagellated stage of *B. canis* have been made by various observers, the most definite statements being those of Breinl and Hindle (1908).   These observers claim to have seen bi-

flagellate forms of *B. canis* in the blood of dogs the day before death. The forms described are 6 to 8 microns in length by 2 to 3 microns in breadth. There is a spherical nucleus with a central karyosome. On one side of the nucleus are two small granules, from each of which a flagellum arises. The organisms resemble flagellates of the genus *Bodo* (Fig. 249). It seems clear that the blood-films must have become contaminated, though it is just possible that the flagellates, if they happened to be present in the intestine of the dog, might invade the blood shortly before death. Such an invasion of the blood by intestinal flagellates has been noted on a number of occasions (see p. 271). Whatever be the explanation of their presence in the films, it is evident that they have no place in the life-cycle of *B. canis*.

Direct Inoculation of Animals.—*B. canis* is readily conveyed from dog
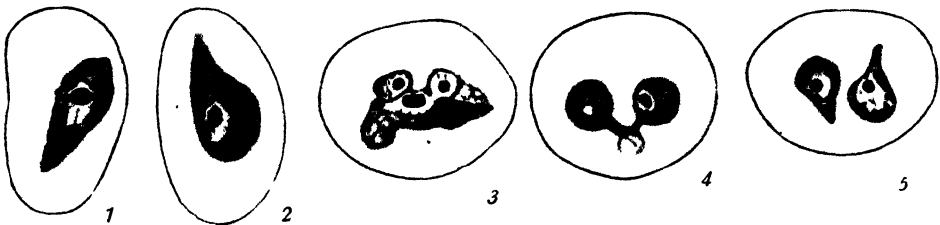
Fig. 419.—*Babesia canis :* Method of Bud Formation, as seen in Wet Fixed Films. (After Schuberg and Reichenow, 1912.)

1. Ordinary pear-shaped form.
2. Pear-shaped form with spherical nucleus with central karyosome and accessory granule.
3. The accessory granule has divided while the karyosome is in process of division.
4. Buds nearly complete, each with karyosome in nucleus and accessory body.
5. Two buds complete. The accessory body is no longer visible.

to dog by inoculation of blood, and this may be effected long after complete clinical recovery and apparent disappearance of parasites from the blood. Nuttall and Graham-Smith, Robertson, and Nocard and Motas attempted to infect numerous animals other than dogs without any result. Nawrotzky (1912) infected dogs by introducing blood into the stomach by means of a tube. Nuttall and Graham-Smith (1909*a*) failed to infect five foxes by inoculating them with infected dog's blood. They report that Dunsbury has informed them of a second failure to infect the jackal, his first attempt having been reported by him (1903). As these nearly related animals cannot be infected, it is evident that *B. canis* is very specific for the dog.

Schilling and Friedrich (1912) inoculated a dog which had suffered from the disease two years previously and produced an infection. In this case there were no data given as to the origin of the two viruses. A similar experiment was made by Ciuca (1913). A dog which had been

infected with a Tonkin virus was subsequently infected with a South African virus. Laveran and Nattan-Larrier (1913) investigated the French and North African strains. The dogs of France were susceptible to both forms, which produced a high rate of mortality. Seven dogs which had recovered from the disease caused by the French virus and were immune to further inoculations were all infected with the North African one. It is concluded that the French and North African parasites are either distinct species or varieties.

Transmission.—Lounsbury (1901, 1904a) was the first to demonstrate experimentally that in South Africa *B. canis* is transmitted by the tick, *Hæmaphysalis leachi*. He showed that if the adult tick fed upon infected animals, the virus passed into the egg and infected the succeeding generation of ticks. The larvæ and nymphs, however, were not infective, as the disease was not transmitted till the adult stage was reached. The disease was later transmitted to dogs in England by Nuttall and Graham-Smith by means of these ticks sent from South Africa. Christophers (1907, 1907b) gave an account of experiments conducted with *Rhipicephalus sanguineus* in India (Fig. 420). He proved that the eggs laid by an adult which had fed on

FIG. 420.—*Rhipicephalus sanguineus* (♀), ONE OF THE TRANSMITTERS OF *Babesia canis* (× 10). (ORIGINAL.)

infected dogs gave rise to larvæ which were not infective, but that the nymphs, and probably the adults resulting, were infective. Specimens of *R. sanguineus* brought to England by James infected English dogs. Similarly, the writer brought to England specimens from Aleppo, which infected a dog six months later. This tick is widely distributed, and is undoubtedly responsible for transmission in Asia, Europe, and North Africa. Brumpt (1919a) proved it to be a vector of *B. canis* of Tunis. Another tick, *Dermacentor reticulatus*, long suspected to be a carrier in Europe, was proved to be a vector by Brumpt (1919) in France. Working with *D. venustus* obtained from North America, Brumpt and Larrousse (1922) fed adult ticks on infected dogs, and later transmitted the infection by means of the offspring raised from eggs laid by these ticks.

The virus taken up by the adult passes through the egg, but infection is not transmitted by the larvæ or nymphs, but only when the adult stage is reached.

The Cycle in the Tick.—This has been studied chiefly by Christophers (1907b) in India (Fig. 421). The blood forms after ingestion by the tick soon leave the host cell, and by increase in size become globular bodies 4 to 5 microns in diameter. These appear to partially divide, and one portion swings round, so that there is produced an elongate body, which
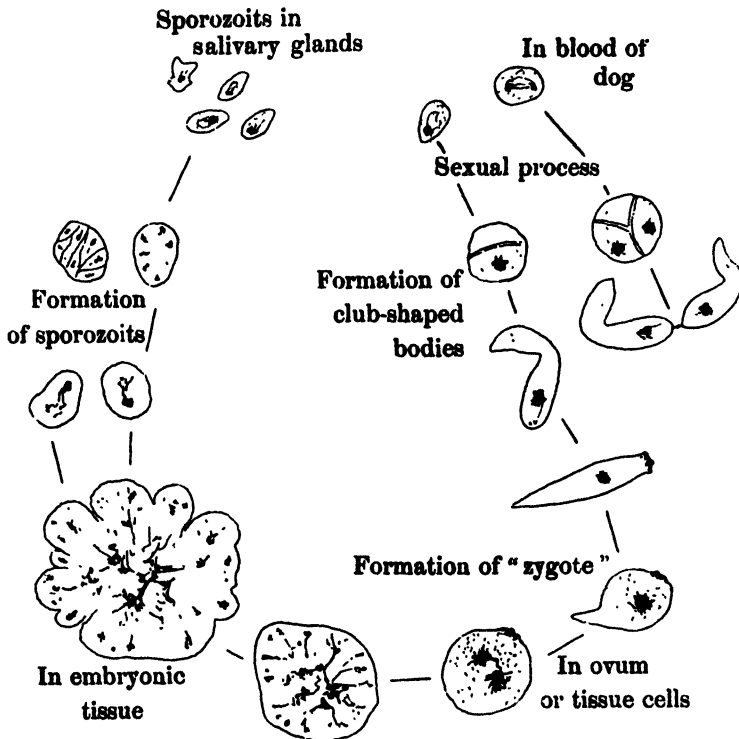
FIG. 421.—*Babesia canis*: DEVELOPMENT IN THE TICK, *Rhipicephalus sanguineus*, ACCORDING TO CHRISTOPHERS ($\times$ ca. 1,500). (AFTER NUTTALL, 1913.)

soon becomes club-shaped. Whether any sexual process is associated with this transformation was not determined. The club-shaped bodies then leave the gut by passing through its wall and enter the tissues of the body. They then become rounded, and increase in size till they may have a diameter of 25 microns. Christophers speaks of these forms as zygotes, a term which implies a previous conjugation, which, however, was not demonstrated.

The nucleus, at first single, multiplies till several are present in the form of a number of masses connected by a plexus of chromatic filaments,

many of which end near the periphery of the parasite in slightly clubbed extremities. Division of this large body into a number of so-called sporoblasts then takes place. Sometimes these appear to be uniform in size, while at others there is considerable irregularity. The nucleus of each multiplies till a large number is present, when segmentation into a corresponding number of sporozoites occurs. The latter are small uninucleate bodies which resemble the small parasites seen in the blood of the dog. They may be pear-shaped, round, star-shaped, or quite irregular in form, and can be found in large numbers in the salivary glands. In larvæ hatched from eggs laid by females which had fed on infected dogs, sporoblasts could be found distributed amongst the tissues, and presumably the sporozoites developed from them ultimately find their way to the salivary glands of the adults. The formation
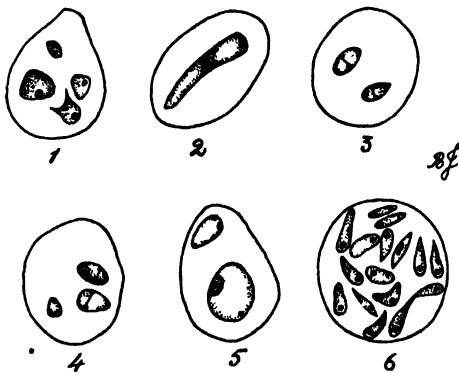
FIG. 422.—*Babesia gibsoni* FROM THE BLOOD OF MADRAS HOUNDS AND JACKALS (× *ca.* 2,000). (AFTER PATTON, 1910.)

of club-shaped bodies and the development of the zygotes, as Christophers terms the growing forms developed from them, were studied in nymphs fed on infected dogs. The sporoblasts become distributed amongst the various developing embryonic tissues, some of which are undoubtedly destined to become the salivary glands of the adults. The developmental process was studied in dried smears, and it is evident that further investigations with more reliable technique are required. It seems quite possible that some of the stages described as zygotes are actually tissue cells of the tick. No one has succeeded in confirming this cycle of development.

Babesia gibsoni (Patton, 1910).—This organism was discovered in the blood of dogs in Madras by Patton (1910), and later in jackals (*Canis aureus*), for the hunting of which the imported dogs were employed (Fig. 422). A further account of the parasite was given by Symonds and Patton (1912). The parasite differs from *B. canis* in being generally smaller in size, and in the absence of the characteristic pairs of pear-shaped forms. Its usual shape is that of a small ring or oval, which occupies not more than an eighth of the diameter of the red cell. There is either a single dot of chromatin or there may be two dots connected by a thread. Occasionally, there occur larger ovoid forms having a length of half the diameter of the cell, and also elongate parasites nearly as long as the diameter of the cell itself. Repro-

duction appears to be by binary fission, and in heavy infections the cell may be crowded with parasites. It was shown by Patton (1910) that dogs which had recovered from infections due to *B. canis* were inoculable with *B. gibsoni*. Nothing definite is known of the transmission, though *Rhipicephalus simus*, which occurs on jackals, is suspected to be the agent.

On account of the absence of typical pear-shaped forms, França (1917) considered *B. gibsoni* should be placed in the genus *Achromaticus*. There does not seem to be sufficient ground for this conclusion. The parasite described by Plimmer (1915) as a *Babesia* from the blood of a wild dog (*Cyon dukhunensis*) of India may possibly be the same as *B. gibsoni*. The writer has seen the parasite in large numbers in the blood of a Colombo dog. The large piroplasma seen by Yakimoff and Schokhor (1917) in wolves in Turkestan is said to be very common, and to correspond morphologically with *B. canis*.
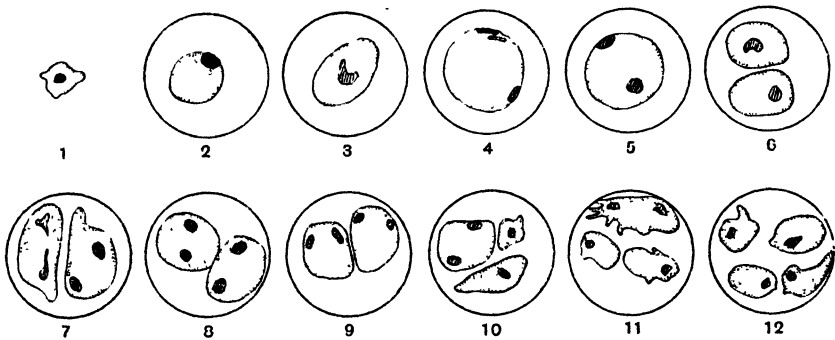
FIG. 423.—*Babesia rossi* (*Rossiella rossi*) FROM THE JACKAL (*Canis adustus*) (× *ca.* 2,000). (AFTER NUTTALL, 1913; FROM *Parasitology*, vol. vi., p. 318.)

Babesia rossi (Nuttall, 1910).—This parasite was seen by Nuttall in blood-films of the East African jackal, *Canis adustus*. It is a large rounded organism, having a diameter up to one-half of that of the red corpuscle (Fig. 423). The nucleus is in the form of a compact body. The parasite occurs in the cells either singly, in pairs, or in fours. Reproduction appears to be effected by simple division. This parasite was placed by Nuttall (1912) in a new genus as *Rossiella rossi*.

Babesia vitalii (Pestana, 1910).—As already pointed out, *B. canis* has not been recorded from America, save for a record by Martinez in Porto Rico. Its place seems to be taken by the parasite discovered by Pestana in dogs in Brazil, which produces a disease similar to the malignant jaundice of the Old World (Fig. 424). The parasites occur in the red cells as round, ovoid, or pear-shaped bodies. The latter are often arranged in pairs which result from double budding, as in the case of *B. canis*. Carini and Maciel

(1914a) and Carini (1915) discovered that in smears of the lungs and kidneys large masses of the parasites occurred within endothelial cells. They interpreted these appearances as being due to a process of schizogony, and note the resemblance to the schizogony which occurs in the case of the toxoplasmas. On account of the supposed double method of multiplication, they placed the parasite in the new genus *Rangelia*. A toxoplasma of dogs is known to occur in Brazil, and it is possible the intracellular forms found in the organs belonged to this parasite, and had no connection with the blood form. It is also possible that the masses in the endothelial cells merely represent phagocyted organisms of the blood type. These two
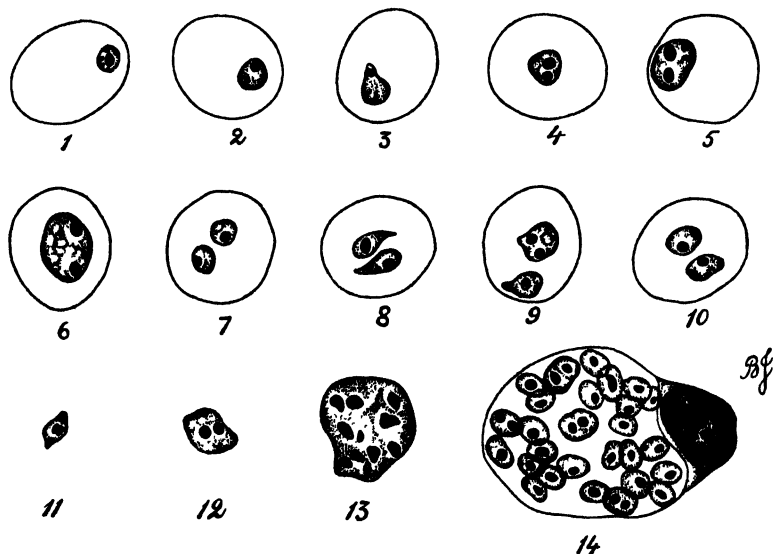
FIG. 424.—*Babesia vitalii (Rangelia vitalii)* OF THE BRAZILIAN DOG (× *ca.* 2,000).
(AFTER CARINI, 1915.)
1-10. Forms in red blood-corpuscles.   11-13. Free forms in internal organs.
14. Completion of schizogony within a cell.

possibilities have certainly not been excluded, so that it seems premature to regard the blood parasite as belonging to a separate genus. Canine piroplasmosis due to *B. canis* has not been recorded from South America, where one would expect it to occur, so that it seems possible that further investigation will show that *B. vitalii* in reality is the same as *B. canis*, which produces the well-known disease of dogs in other parts of the world.

BABESIA OF SMALL MAMMALS.

The first of the unpigmented parasites of the red cells of small mammals to be described was the form named *Achromaticus vesperuginis* by Dionisi (1899), who discovered it in 1898, together with a pigmented parasite,

in the bat, *Vesperugo noctula*, in Italy. Since that time a number of unpigmented parasites have been described from small mammals. Some of these are comparatively large forms, while others are small, and resemble either *B. equi* or *B. mutans.* They have been placed in various genera, but it seems better to retain them in the genus *Babesia.*

Babesia vesperuginis (Dionisi, 1899).—As just remarked, this parasite was first seen by Dionisi in *V. noctula.* According to Schingareff (1906), it was seen in the same bat by Berestneff in 1903, as also by Galli-Valerio (1905), and Neumann (1909*a*). Kisskalt (1905), and Coles (1914) observed it in *V. pipistrellus*, and Gonder (1906) in *V. kuhli.* Yakimoff, Stolnikoff and Kohl-Yakimoff (1912) gave an account of the organism as seen in an unidentified bat of Turkestan. The parasite as it occurs in the blood shows a great variation in form and size (Plate XVI., 28-32, p. 974). It may be sickle-shaped, round, piriform, semilunar, or quite irregularly amœboid. Some of the parasites occur as band forms stretching across the red cell. The majority of the forms, however, are very much like those which appear in infections of *B. canis*, and this applies specially to the pear-shaped individuals. Round and sickle forms were seen free in the plasma, and the latter may be applied to the margins of the red cells, as occurs in the case of the marginal forms of the human malarial parasites. Some of the sickle forms are barely 2 microns in length, while others are larger and may reach a length of half the diameter of the red cell. Within the cells all forms occur. The round forms vary in diameter up to a third of that of the cell. In the largest ones nuclear multiplication has taken place till four are present. Four pear-shaped individuals are then formed, and these may be arranged as a cross or in the form of a fan. Coles (1914) remarks on the resemblance of the pear-shaped forms to those seen in the typical piroplasmata, and notes also the division of the organism into four pear-shaped individuals, which are arranged in the form of a cross, as occurs in the case of *B. equi* of horses.

Neumann (1909*a*) observed certain structures in the body of mites (*Pteroptus vespertilionis*) taken off infected bats, which he believed represented developmental forms of *B. vespertilionis.*

In a later paper, Yakimoff Stolnikoff, and Kohl-Yakimoff (1916) describe another unpigmented parasite from a bat of Turkestan. It resembles *B. vesperuginis* in the presence of round, ovoid, amœboid, and pear-shaped forms. In addition, there occur larger forms, which nearly fill the red cell and possess several nuclei. The observers consider these to be of the nature of schizonts, and suggest that the parasite forms a connecting link between the pigmented plasmodia and the unpigmented piroplasmata. They proposed to name the organism *Plasmodium achromaticum* without having demonstrated that they

were not actually dealing with hitherto undescribed stages of *B. ves-peruginis.*

Babesia avicularis Wenyon, 1909.—This is an unpigmented parasite which was discovered by the writer in the striped zebra mouse (*Arvicanthis zebra*) of the Sudan (Fig. 425). It occurs as round, ovoid, or angular masses of cytoplasm with a single chromatin body. These forms have a diameter of about a sixth to a third of that of the cell. Some of the parasites are very irregular in shape, and resemble the amœboid forms of *Plasmodium falciparum* in human blood. A somewhat similar, if not identical, form was seen by Bruce *et al.* (1911*g*) in a white mouse in Uganda. The animal originally came from Naples, and had been inoculated with blood from another mouse which had been infected with spirochætes from a bush buck. There were present in the red cells pear-shaped forms occurring singly or in
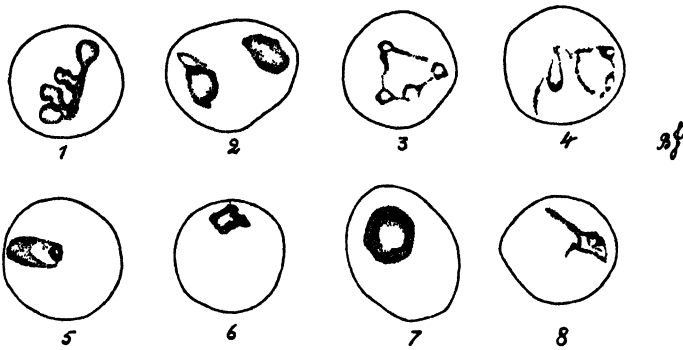
FIG. 425.—*Babesia avicularis* OF THE ZEBRA MOUSE, *Arvicanthis zebra* (*Lemniocomys zebra*) OF THE SUDAN (× 3,000). (AFTER WENYON, 1909.)

couples, and measuring about 5 microns in length, round or irregularly shaped forms, and others showing an indication of division into four. What was possibly the same parasite was again seen by Bruce *et al.* (1915) in the edible rat of Nyasaland.

Babesia quadrigemina (Nicolle, 1907).—This parasite was discovered by Nicolle (1907) in the gondi, *Ctenodactylus gundi*, of Tunis (Fig. 426). The organism occurs as rounded forms which have a diameter of a quarter to a third of that of the red cell, as ovoid and also pear-shaped forms. Multiplication takes place by a division of the nucleus into four, and the formation of four pear-shaped daughter individuals which are arranged either in a cross or fan-like manner. The nucleus consists of a chromatin body, near which is a smaller granule. Both these bodies probably belong to the nucleus, the structure of which cannot be properly detected in dried films. Nuttall (1908), who studied the organism in films sent him by Nicolle,

speaks of the nucleus as showing distinct " binuclearity." On account of its method of reproduction he founded a new genus for the parasite, which he named *Nicollia quadrigemina*. The organism is a typical piroplasma, and there appears to be no adequate reason for its inclusion in any genus other than *Babesia*. It bears a striking resemblance to *Plasmodium minasense* (Plate XVII., 6-15, p. 982).

Under the name of *Piroplasma muris*, Fantham (1906) described a parasite which he found in the blood of a white rat which had died in a London laboratory. It is remarkable that, in spite of the fact that blood-films of white rats are subject to constant examination in the course of laboratory work, no one has rediscovered the organism. Nuttall and Graham-Smith (1908), who examined a film sent them by Fantham, remark that in this film they saw piriform intracorpuscular parasites singly and in pairs.
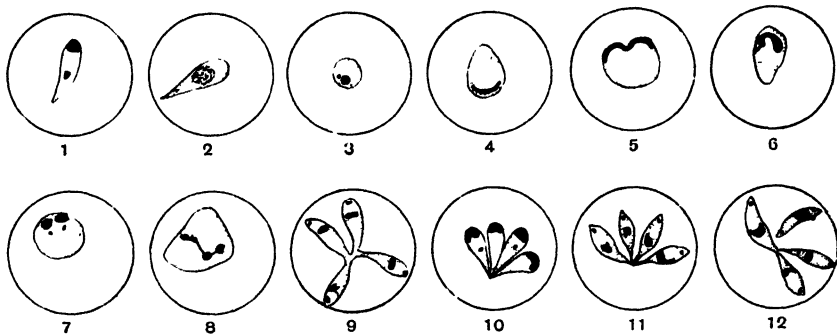
FIG. 426.—*Babesia quadrigemina* (*Nicollia quadrigemina*) FROM THE BLOOD OF THE GONDI (*Ctenodactylus gundi*) (× ca. 3,000). (AFTER NUTTALL, 1913; FROM *Parasitology*, vol. vi., p. 318.)

Dschunkowsky and Luhs (1909) discovered a piroplasma (*B. leporis*) in the Transcaucasian hare. *Babesia herpestidis* was seen by França (1908) in the mongoose, *Herpestes ichneumon*, in Portugal, while an unnamed form was mentioned by Patton (1910) as occurring in the Indian mongoose (*H. mungo*). Another, found by Bédier (1924) in the African mongoose, *H. calera*, was named by him *Nuttalia legeri*. Yakimoff (1910a) discovered one of these parasites, which he named *Piroplasma ninense*, in the hedgehog (*Erinaceus europœus*) of Russia, while Galli-Valerio (1911) observed one (*B. weissi*) in *E. algirus* of Tunis. Coles (1914), under the name of *Nuttallia muris*, described a parasite of the English field mouse, *Mus sylvaticus*, and another as *N. microti* of the water vole, *Microtus amphibius*. Macfie (1915) described and figured a similar parasite from *Mus decumanus* (*Rattus norvegicus*) of West Africa. The organism, to which he gave the name *Nuttallia decumani*, was a typical *Babesia* showing single and double

forms, as also radially arranged groups of four (Fig. 427). He also recorded the presence of " piroplasma-like " bodies in a guinea-pig. Leger and Bédier (1923a) have recorded as *Nuttallia golundæ* a small parasite of the rodent, *Golunda campanæ*, of Senegal. França (1909) observed a parasite of the field mouse, *Microtus incertus*, in Portugal. Though most of the forms present resembled those seen in the infections just described, there were some pear-shaped parasites which extended from one side of the cell to the other. On account of these large forms, França established a new genus, *Smithia*, and named his parasite *S. microti*. Galli-Valerio (1914) placed in this genus, under the name of *S. talpæ*, a parasite discovered by him in the European mole, *Talpa europæa*. Yakimoff and Saphronowitsch (1917) gave the name of *Theileria rossica* to a parasite seen by them in several field voles near Kars in Transcaucasia. No schizogony forms characteristic of this genus were seen, so that it cannot be retained in the genus *Theileria*. Franchini (1924a) has given the name
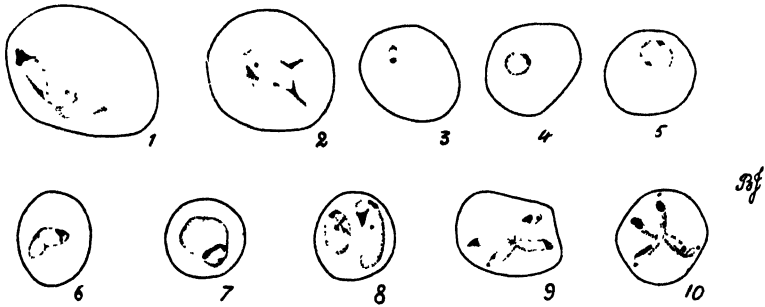
FIG. 427.—*Babesia decumani* FROM THE BLOOD OF THE BROWN RAT, *Rattus norvegicus* OF NIGERIA (× 2,000). (AFTER MACFIE, 1915.)

Nuttallia myoxi to a parasite of *Myoxus avellanarius*, the dormouse, in Italy. It is very similar to *N. decumani*, described by Macfie (1915). Another form discovered by Leger and Mouzels (1917) in the three-toed sloth (*Bradypus tridactylus*) was considered to be a *Theileria*, and named *T. brimonti*, from the character of the blood forms alone. A small piroplasma-like organism was encountered by A. and M. Leger (1920) in the red cells of the civet (*Viverra civetta*) of Senegal. They consider it as allied to *B. herpestidis* of the mongoose, and give it the name *Nuttallia civettæ*. Leger, M., and Bédier (1922a) record the presence of a small round piroplasm, which they name *Nuttallia bauryi*, in the fox, *Fennecus dorsalis* of the French Sudan. The parasite had a diameter of 1·25 to 1·5 microns. Yakimoff, Kohl-Yakimoff and Korssak (1910) mention a piroplasma of the Russian fox. What is probably the same parasite is referred to by Yakimoff and Schokhor (1917), who without any evidence of the existence of schizogony stages placed it in the genus *Theileria*.

BABESIA OF MONKEYS.

Babesia pitheci (Ross, 1905).—This parasite, to which he gave the name *Piroplasma pitheci*, was discovered by Ross, P. H. (1905), in a species of *Cercopithecus* in Uganda. The organism was later studied by Nuttall and Graham-Smith (1908) in films sent them by Ross. According to them, the organism is a typical piroplasma of the *B. canis* or *B. bigemina* type, as Ross had stated in the first place. Reproduction by budding in the characteristic manner occurs. The parasites are either round, oval, or irregular in outline, while pear-shaped forms occur either singly or arranged in pairs. Smears of the organs showed a larger number of parasites than the peripheral blood. As many as sixteen parasites were seen in a single cell. Castellani and Chalmers (1910), under the name *Babesia cellii* Castellani and Chalmers, 1908, mention the occurrence of a piroplasma of the monkey, *Macacus pileatus*. They state that it occurs as bacillary and pear-shaped forms, lying side by side in the erythrocytes. In 1919 they substituted the name *Theileria cellii* Castellani and Chalmers, 1910, without giving any further details.

BABESIA OF ANTELOPES AND OTHER ANIMALS.

A number of small piroplasmata have been described from antelopes and deer. A form resembling *B. equi* was seen by Denier (1907) in *Cervus aristotelis* in Annam, and Nuttall (1913) mentions similar forms seen by him in blood-films of deer and reindeer sent him by Marzinowsky from Russia. Kerzelli (1909) had reported a similar form from the reindeer. Ollwig and Manteufel (1912) state that Kudicke observed parasites of this type in the zebra. A small form had already been seen in this animal by Ross, P. H. (1907). in Uganda, and by Theiler (1907a). Parasites of the *B. mutans* type have been seen by various writers. Thus, *B. cervi (syn. B. damœ)* was seen by Bettencourt, França and Borges (1907) in *Cervus dama* of Portugal, an unnamed species by Bettencourt and Borges (1909) in *Cephalophus grimmi*, another by Patton (1910) in *Cervus axis* of India, and another in *Boselaphus* sp. by Lichtenheld (1911) in East Africa. Ross, P. H. (1911). reported the finding of small forms of the *B. mutans* type in various game, including *Bubalus cokei, Æpyceros melampus, Tragelaphus scriptus, Oryx beisa, O. callotis, Gazella thomsoni*, and the zebra, *Equus burchelli granti*. *B. hippotragi* was noted by Todd and Wolbach (1912) in *Hippotragus equinus* of the Gambia, *B. stordii* by França (1912a) in *Gazella grantii* of Abyssinia, and an unnamed form by Rodhain (1916) in *Cobus defassa* of the Belgian Congo. Carpano (1913a) states that he called attention in 1911 to a form in the gazelle which appears to be identical with *B. stordii*. Various observers have

seen small piroplasmata in antelopes in Africa without recording names of the hosts. Leger, M., and Bédier (1922a) state that they have seen a small type of piroplasma in a young lion in the French Sudan, while Schein (1923) records one from *Cervulus muntjac* of Annam. Yakimoff, Kohl-Yakimoff and Korssak (1910) mention the occurrence of piroplasmata in the fox, Chinese yak, and bear in Russia. The occurrence of *B. bigemina* in white-tailed deer in Panama, as noted by Clark (1918), has been referred to above (p. 996).

Cultivation of Babesia.

Many attempts have been made to cultivate the various species of *Babesia*. In the earlier investigations, infected blood was merely diluted with saline and kept at various temperatures. It was shown that parasites remained virulent for many days, especially if kept at low temperatures. The extensive and careful investigations of Nuttall and his co-workers from 1908 onwards have shown that various species of piroplasmata will continue their development *in vitro* on the warm stage and that their movements and method of multiplication can be studied in this manner. The parasites behaved in a similar manner in cultures, but degeneration quickly took place. Nuttall and Graham-Smith (1908) noted, however, that increase in size of *B. canis* occurred, and that forms with long, spiky, radiating pseudopodia appeared. These resembled in some respects the amœboid forms described by Koch as occurring in ticks which had ingested *B. bigemina* of cattle (Fig. 410). Miyajima (1907) claimed to have obtained a culture of a small piroplasma of cattle. In his cultures he obtained trypanosomes and developmental forms of these, and concluded that he had demonstrated their origin from the piroplasmata. There can be no doubt that Miyajima had isolated *Trypanosoma theileri*, which is easily cultivated from the blood of cattle. The publication by Bass and Johns (1912) of a method of obtaining cultures of the malarial parasites naturally led observers to attempt this method for the cultivation of the piroplasmata. Knuth and Richters (1913), Ziemann (1913, 1914), and Thomson and Fantham (1913, 1914) seem to have been the first to attempt this in the case of *B. canis*. The method employed was essentially the same as that which has been described above (see p. 967). The development takes place, however, with less regularity than in the case of the malarial parasites, and though Ziemann obtained subcultures these were not satisfactory. In the primary culture, multiplication of the parasites occurs, and a marked increase in their numbers takes place, so that red cells may be seen packed with parasites, though such heavily infected cells were not present in the blood of the dog from which the culture was made. There

is nothing remarkable about the culture forms, which resemble those seen in the blood of the dog. In old cultures, many abnormal degenerating forms occur. Ziemann showed that a culture was virulent for dogs up to the twentieth day. Vrijburg (1913) attempted culture of *B. bigemina* in the same way, but did not obtain satisfactory results.

2. *Family:* THEILERIIDÆ.

This family has been defined as including unpigmented parasites of red blood-corpuscles, which reproduce by schizogony within endothelial cells of the capillaries of the internal organs. Certain forms produced in the endothelial cells enter the red cells, and appear in the peripheral blood. Contrary to what occurs amongst the various species of *Babesia*, these blood forms do not multiply in the red cells. On this account the blood is not infective when inoculated to healthy animals unless endothelial cells containing schizonts happen to be present. The family includes the single genus *Theileria*, the best-known species of which is *T. parva*, the cause of East Coast fever of cattle in Africa and elsewhere. The parasite, which Dschunkowsky and Luhs (1904) discovered in cattle in South Russia, and which they named *Piroplasma annulatum*, may be the same species. Their discovery (1909) of schizonts in the organs proves that it belongs to the genus *Theileria*. According to Mason (1922), Egyptian cattle fever is the same disease, but Sergent, Ed., and his co-workers (1924) believe the disease of North Africa is caused by a distinct parasite *T. dispar*. These two species may, however, be merely races of *T. parva*. Species of *Theileria* have also been recorded from the sheep and goat, and *Echidna* in Australia.

THEILERIA OF CATTLE.

Theileria parva (Theiler, 1904).—This parasite, which was first seen by Koch (1898), who considered it to be a stage of *Babesia bigemina*, produces a serious disease of cattle, East Coast fever, which differs from that produced by *B. bigemina* in that hæmoglobinuria, jaundice, and progressive anæmia are absent. The organism was named *Piroplasma parvum* by Theiler (1904), and was placed in a new genus, *Theileria*, by Bettencourt, França and Borges (1907). Stephens and Christophers (1903*b*) proposed the name *P.* [*Kochi*] for the parasite of "African Coast Fever," so that *Theileria kochi* may be the correct name.

The disease occurs chiefly in Africa, and has been reported from Rhodesia, Transvaal, Uganda, East Africa, and North Africa. West Africa and the Belgian Congo appear to be free from the disease. Outside Africa it has been noted in Transcaucasia by Dschunkowsky and Luhs,

and in Macedonia and India.   According to Koch, the zebus (*Bos indicus*) imported to East Africa from Madagascar, were frequently immune to the disease, a fact which suggests its occurrence in that island.

Morphology.—The morphology and life-history of *T. parva* was studied by Gonder (1910*a*, 1911, 1911*b*), but, unfortunately, his account was obscured by such theoretical bias that it is difficult to separate fact from theory.   He was justified, however, in his conclusion that the bodies (Koch's blue bodies) which occur in the spleen, liver, kidney, lymphatic glands, intestinal mucosa, and other organs represent schizonts.   These structures may be discovered by puncture of the enlarged glands.   In dried smears stained by Romanowsky stains, they appear as blue masses of cytoplasm containing a varying number of red chromatin dots
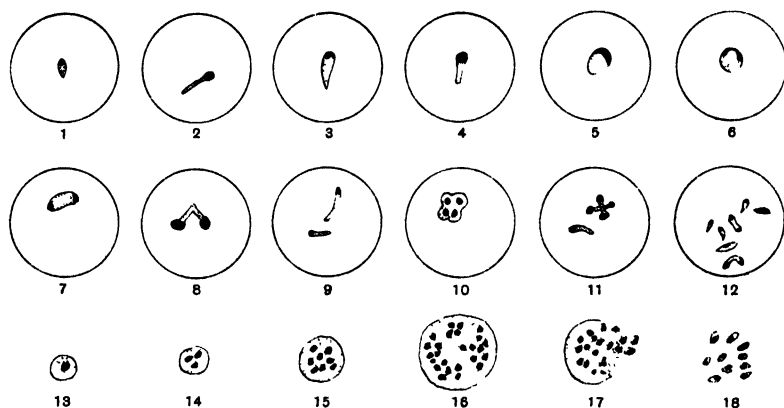
FIG. 428.—*Theileria parva* OF EAST COAST FEVER OF CATTLE (× *ca.* 2,500).
(AFTER NUTTALL, 1913; FROM *Parasitology*, vol. vi., p. 315.)

1-12. Forms in red blood-corpuscles.
13-18. Schizonts (Koch's blue bodies) from the spleen.

(Plate XVIII., 36-38, p. 986, and Fig. 428).   They vary in size from 3 to 10 microns, and in films are seen either free or within cells of the endothelial type.   Their extracellular position is probably due to the breaking down of cells in the process of film making.   In sections they are always intra-cellular.   Koch's blue bodies or schizonts are usually confined to the lymphatic glands, spleen, or other organs, where they occur in the endo-thelial cells of the capillaries.   Occasionally they are found in endothelial cells in the peripheral blood.   Their presence here accounts for the occa-sional positive results obtained by blood inoculation of healthy animals. When fully formed, they break up into numbers of minute bodies, which either enter other cells to grow and reproduce by schizogony again, or they penetrate the red blood-corpuscles, in which they are found in ordinary

blood-films. They are here seen as minute bodies 1 to 2 microns in diameter when round. Some of the forms are ovoid in shape, others are rod-like, while some are pear-shaped or comma-shaped. Though they may sometimes be seen in pairs in the red cells, or occasionally in fours as in the cross form, it is doubtful if these represent division stages, as they do in the case of *B. mutans*, the morphological resemblance to which may be very striking.

The organism in the blood was very carefully studied by Nuttall, Fantham and Porter (1909) in infections produced in England by means of ticks, *Rhipicephalus evertsi*, imported from South Africa (Fig. 428). The minute parasites were seen to move about in the red cells, and to undergo changes in shape. Actual division was never observed to take place in the living condition, though in stained films parasites which might be interpreted as in process of division were sometimes seen.

The blood forms first make their appearance a few days after the first symptoms show themselves, and they increase rapidly in number till 80 to 90 per cent. of the corpuscles are affected. Such heavy infections are not noted in the case of *B. mutans*. Multiple infection of individual cells is of common occurrence. As already remarked, inoculation of blood will not, as a rule, convey the infection, so that it is presumed that the blood forms represent gametocytes which are destined to develop in the tick.

If animals recover from the disease, the parasites disappear from the blood, and this disappearance is absolute, for ticks can no longer be infected from them. In this respect, again, *T. parva* differs from the species of *Babesia*, which, though disappearing microscopically, are still present for years after clinical recovery, as proved by the infectivity of blood on direct inoculation to other cattle, and by the fact that ticks may still infect themselves.

Symptoms and Pathology.—Theiler (1904) was the first to demonstrate that East Coast fever was a distinct disease, and that cattle which had recovered from hæmoglobinuric fever due to *Babesia* were not immune to it. He noted also that it could not be conveyed by blood inoculation, and that the marked anæmia, jaundice, and hæmoglobinuria so characteristic of red-water fever did not occur. About ten to twenty days after exposure to infection the disease commences with fever, which continues till death or recovery takes place. All the superficial lymphatic glands become hypertrophied. They attain their maximum development in about a week, after which they gradually diminish in size. The animals remain in good condition and feed well till the end stages of the illness are reached. The mortality rate is high, and may be as much as 80 to 90 per cent. of those affected. Calves appear to be slightly more tolerant than adults, and the mortality rate is lower amongst animals which are well looked after

than in those that are primarily in poor condition. A common feature of infections with *T. parva* is that old latent infections of *B. bigemina* or *B. mutans* may be stimulated, and the symptoms associated with these forms appear. On this account, some confusion has arisen as to the symptoms which are actually due to *T. parva* and to the character of the parasite as it is found in the blood. Animals which have died of the disease show œdema and swelling of the subcutaneous tissues and intestinal mucosa. Hæmorrhagic patches occur in the mucosæ and on the serous membranes. The spleen does not appear to be much enlarged.

Inoculation of Animals.—Koch (1903) stated that he had transmitted the disease to cattle by two successive intraperitoneal inoculations of large quantities of blood, while Tartakowsky (1905) made a similar claim for the parasite described as *Piroplasma annulatum* by Dschunkowsky and Luhs (1904). In a later paper (1909) the latter observers recorded the presence of Koch's blue bodies in the organs of the infected animals. Carpano (1912, 1915) likewise transmitted the disease by blood inoculation in Tripoli, and noted the presence of Koch's blue bodies in the peripheral blood in severe cases of the disease. Donatien, Plantureux, Rossi and Esperandieu (1923) have confirmed these observations in Algeria. It seems evident, therefore, that the schizonts occur in largest numbers in the lymphatic glands and spleen, but occasionally appear in the peri-

FIG. 429.—*Rhipicephalus evertsi* (♀), A TRANSMITTER OF *Theileria parva*, *Babesia equi*, AND *B. mutans* (× 10). (ORIGINAL.)

pheral blood. Theiler and Stockman and Nuttall, quoted by Nuttall, Fantham and Porter (1909), failed to obtain infection by subcutaneous inoculation of spleen and bone marrow containing the schizonts. Koch (1903) likewise failed to produce the disease by both subcutaneous and intravenous inoculations, but Meyer (1909) claims to have succeeded by inoculating or transplanting portions of spleen into the peritoneal cavity.

Transmission.—Lounsbury (1902-1904) was the first to prove that East Coast fever was transmitted by the tick *Rhipicephalus appendiculatus*. This observation was confirmed by Theiler (1904*b*, 1905), and extended to *R. simus*. Lounsbury (1906) succeeded in conveying the disease by means of *R. evertsi*, *R. nitens*, and *R. capensis* (Fig. 429). With the exception of *R. evertsi*, which has two hosts, these ticks live on separate hosts in the larval, nymphal, and adult stages. The virus taken

up at one stage is transmitted by the next. The adult tick does not transmit the virus through the egg to the larvæ, as occurs in the case of the various species of *Babesia*. Nuttall and Graham-Smith (1909) produced the disease experimentally in cattle in England by means of ticks (*R. evertsi*) sent from South Africa. The factors governing the infectivity of ticks were studied by Nuttall and Hindle (1913). It was shown by them that ticks still remain infective after feeding, for the disease was transmitted to calves by ticks which had fed for three days on rabbits. It was noted, however, that infection was not conveyed unless the ticks remained on the host for more than two days. Calves did not become infected if the ticks were allowed to remain for only two days. That this was not a question of temperature alone was proved by keeping ticks at 37° C. for two days before they were allowed to fix on calves. Transmission still did not occur unless more than two days were allowed to elapse before the ticks were removed.

As regards the transmission of the parasite *Theileria annulata*, which is possibly only a race of *T. parva*, Dschunkowsky and Luhs stated that the carrier was the tick *Margaropus annulatus*, and that the virus passed through the egg to the larva, a feature which does not occur in the case of East Coast fever. These transmission experiments are not sufficiently conclusive, as it is not certain that the cattle used were not infected with species of both *Babesia* and *Theileria*. In Egypt, Mason (1922) notes that the ticks found associated with the disease are *Margaropus annulatus*, *Hyalomma ægyptium*, and *Boophilus australis*, but it is not definitely known which is responsible for transmission.

Cycle in the Tick.—Gonder studied the development of *R. appendiculatus*, but his observations were influenced by a desire to bring the parasite into line with Schaudinn's views on the development of halteridium, with the result that not only are parthenogenetic and other methods of reproduction described, but the flagellate nature of the organism is constantly emphasized. Mature gametocytes leave the red cells and become free in the gut of the tick, where they can be distinguished as male and female forms, the former motile and the latter stationary. After a nuclear reduction a male form is said to unite with a female, when a further reduction of each nucleus takes place. The resulting zygote becomes an oökinete, which penetrates the wall of the gut and makes its way to the salivary glands. Here it increases in size while nuclear multiplication takes place. Finally, a large number of sporozoites is produced, and these are inoculated by the tick when it feeds. Whether this account, even in its broad outline, is correct or not further research alone will show.

Reservoir Hosts.—The possibility of the occurrence of *Theileria parva* in the game of countries in which East Coast fever is indigenous was first raised by Ross, P. H. (1911), who found small parasites in the red blood-corpuscles, and what he regarded as an undoubted Koch's blue body in a smear of the liver of Coke's hartebeest (*Bubalus cokei*). Lichtenheld (1911) also claims to have found similar bodies in a kidney infarct of an eland. Montgomery (1913) likewise observed them in a bastard hartebeest. He informs the writer that the small parasites which he discovered in Grant's gazelle, and which França (1912a) named *T. stordii*, were present in such large numbers in the blood-films that the infection resembled one of *T. parva* rather than *Babesia mutans*. It is thus possible that antelopes may act as reservoirs for *T. parva*, and the same statement may be made of *B. mutans*. At present, however, there is no experimental evidence either for or against this view, but experience appears to indicate that in game country cattle are, if anything, less liable to East Coast fever than in areas free from game.

Other Diseases of Cattle possibly due to Species of Theileria.

Dschunkowsky and Luhs (1904 and 1909) described a disease of cattle in Transcaucasia which resembled in many respects East Coast fever. In the blood the parasites were mostly in the form of minute rings, though ovoid and rod-shaped forms also occurred. Koch's blue bodies were found in smears of the lymphatic glands, kidneys, and spleen. The organism was named *Piroplasma annulatum*, but the presence of schizonts shows that it belongs to the genus *Theileria*. Dschunkowsky and Luhs (1904) stated that the parasite was not inoculable by means of infected blood, and this observation led Koch to express the opinion that the disease was actually East Coast fever. Tartakowsky (1905), however, stated that such inoculation was possible, but it is far from clear that in these cases there was not a mixed infection with *Babesia mutans*. Mason (1922) describes a disease known as Egyptian fever of cattle, which had previously been noted by Bitter (1905) and himself (1915). It occurs in an acute, subacute, and chronic form, and the parasites in the blood correspond with those described by Dschunkowsky and Luhs. Mason states that he has seen the same disease in Cyprus and in cattle imported to Egypt from the Sudan. He concludes that the piroplasmosis of cattle in Morocco described by Velu (1921) is of the same type. Mason found Koch's blue bodies in the liver, lymphatic glands, ulcers in the abomasum, in the small pin-point infarcts in the kidneys, and in the hæmorrhagic infarcts in the spleen. The disease differs from East Coast fever in that cattle, if they survive the first attack, become chronic carriers of the virus, and acute or sub-acute attacks which may prove fatal are induced by exhaustion following overwork and fatigue. Mason is of the opinion that the disease is distinct from East Coast fever, and is due to a *Theileria*, which he regards as probably identical with the form described by Dschunkowsky and Luhs, and which would become *T. annulata* (Dschunkowsky and Luhs, 1904). In addition to cattle, buffaloes are liable to infection.

The question of *T. annulata* has, however, been investigated by Donatien, Plantureux, Rossi and Esperandieu (1923), and Sergent (1923) in **Algeria.** Ducloux (1905) in **Tunis,** and Soulié and Roig (1908, 1908a) in **Algeria,**

described a disease of cattle which resembled East Coast fever. They thought that the minute piroplasma which occurred in the blood resembled *P. annulatum* of Dschunkowsky and Luhs. Sergent, Ed. and Et., and Lhéritier (1919a) recorded the presence of Koch's blue bodies in the lymphatic glands of jaundiced cattle in Algeria, and Donatien and his co-workers (1923) have also discovered the presence of these bodies in sick cattle in the same country. Though usually confined to the lymphatic glands and other organs, they occasionally occur in the peripheral blood within large mononuclear cells, or even free in the plasma. In such cases it was demonstrated that the disease could be reproduced in other animals by blood inoculation, as previously claimed by Koch (1903), Tartakowsky (1905), and Carpano (1912, 1915). Sergent (1923) pointed out that the parasite described by Dschunkowsky and Luhs as *P. annulatum* did not differ in any way from that found in cattle in Algeria or in East Africa. He concluded that only one parasite, *T. parva*, was responsible for the disease of cattle in East Africa, North Africa, and Transcaucasia. He emphasized the fact that it was often associated with *B. mutans* and *Anaplasma*, and that the occurrence of jaundice or even hæmoglobinuria, which do not occur in typical pure infections with *T. parva*, might be accounted for by the mixture of infections. The Egyptian disease may be identical with that of Algeria, but that these are East Coast fever is doubtful, since Sergent and his co-workers (1924) recognize a distinct parasite, *Theileria dispar*, as the cause of the Algerian disease.

Brumpt (1923) describes a series of experiments with a Tunisian virus which appeared to be undoubtedly *B. mutans*. During a series of passages by direct inoculation of blood made in France it was found that some of the animals acquired an intense and fatal infection associated not only with the presence in the red blood-corpuscles of enormous numbers of parasites of the usual type, but also with the occurrence of schizonts in the peripheral blood and internal organs. These schizonts were very similar to Koch's blue bodies, and Brumpt reaches the conclusion that the parasite usually called *B. mutans* is in reality a *Theileria*, and that its correct name is *T. mutans*. Usually the infections are mild and schizonts are not apparent, but in intense infections they occur in large numbers and the true nature of the parasite is revealed. The parasite *T. mutans* differs from *T. parva* in certain respects, though the general appearance of the blood forms and schizonts is very similar. The ovoid, annular, bacillary, comma-shaped, and cross forms appear in different proportions in the two infections. Both binary and quarternary division forms occur in both infections. The schizonts of *T. mutans* are larger than those of *T. parva*, while the nuclei of the former tend to be ovoid in shape and those of the latter spherical. There is, however, a profound difference in virulence. Infections in adult animals due to *T. mutans* prove fatal in only 5 to 10 per cent. of cases, while in those due to *T. parva* the mortality reaches 95 to 100 per cent. Calves are much less susceptible, though the same difference is noted. Animals which have recovered from *T. parva* infections no longer harbour parasites, and are completely immune, whereas those infected with *T. mutans* constantly harbour the parasites, and are liable to relapses during the rest of their lives. A small dose of blood from animals harbouring *T. mutans* injected into healthy animals conveys the infection, whereas large doses of blood from animals infected with *T. parva* does not do so, though injection of material from the organs containing schizonts conveys infection. Velu (1923), working in Morocco, has also noted that the small piroplasma of cattle is readily inoculable from animal to animal. In those cases in which a mild infection is produced the parasites occur only in the blood, and schizonts appear to be absent from the organs. When a virulent infection is produced, the schizonts are present. The animals remain carriers for

long periods after recovery. Velu's observations are thus a direct confirmation of those of Brumpt. Doyle (1924) in Cyprus and Edwards (1925) in India have come to the same conclusion after finding blue bodies in cattle infected with what appeared to be *B. mutans*. They are present, however, in small numbers only, a feature which distinguishes the infection from true East Coast fever. On the other hand, Sergent, Ed., and his co-workers (1924) believe that in Algeria two small parasites occur, one of which is *B. mutans* (they employ the name *Gonderia mutans*), and the other a *Theileria*, to which they give the new name *T. dispar* without considering *T. annulata*. They come to the conclusion that *B. mutans* never produces schizonts, and they disagree with Brumpt that the two parasites are one. *T. dispar* is said to differ from *T. parva* in that it is readily inoculable by means of blood, and that it gives rise to anæmia and splenomegaly.

Brumpt (1924) reviews the recent work on the small piroplasmata of cattle, and concludes that the following four species of *Theileria* occur which differ from one another biologically:

T. mutans. — Non-pathogenic, transmissible by direct blood inoculation, persistence of parasites in the blood for long periods (no immunity).

T. annulata.—Sometimes pathogenic, transmissible by direct blood inoculation, persistence of parasites in the blood for long periods, at any time during which a fatal relapse may occur (no immunity).

T. dispar. — Pathogenic, transmissible by direct blood inoculation, no persistence of parasites after recovery (complete immunity).

T. parva.—Highly pathogenic, not transmissible by direct blood inoculation, no persistence of parasites after recovery (complete immunity).

It is admitted that these species can hardly be differentiated morphologically, and that they represent a series the individuals of which merge into one another by gradations. Thus, there are types of infection with *T. annulata* which cannot be distinguished from those due to *T. mutans*. Koch's blue bodies occur very rarely in *T. mutans* infections in which reproduction of the parasites in the red blood-corpuscles occurs. At the other extreme is *T. parva*, in infections with which Koch's blue bodies are constantly present, while there are no multiplying forms in the red blood-corpuscles.

THEILERIA OF SHEEP, GOATS, AND OTHER ANIMALS.

Though many parasites have been placed in the genus *Theileria*, in only two instances, apart from *T. parva* of cattle and the other species found in these animals, if they are authentic, has the presence of schizonts been observed. Accordingly all the forms have been considered above as belonging to the genus *Babesia* unless the presence of schizonts like those occurring in the development of *T. parva* has been definitely recorded.

Theileria hirci Dschunkowsky and Urodschevich, 1924.—The occurrence in sheep and goats of a small parasite resembling *B. mutans* has been referred to above (p. 1007). Though the name *T. ovis* was suggested by various observers for a parasite of sheep, they produced no evidence that reproduction by schizogony occurred, so that there is no alternative but to regard the parasites as belonging to the genus *Babesia* and allied to *B. mutans*. Mason (1915 and 1916) has, however, described what appears to be a true *Theileria* from Egyptian and Sudanese

sheep. The blood forms resembled those of *T. parva* of cattle, while Koch's blue bodies (schizonts) were found in the lymphatic glands, and also in the spleen, which was enlarged to three or four times its normal size. As the name *T. ovis* was suggested for the small parasite belonging to the genus *Babesia*, it is no longer available for the true *Theileria* of sheep, though Mason uses it in this sense. Working in Serbia, Dschunkowsky and Urodschevich (1924) encountered a small parasite in the red blood-cells of goats. In the peripheral blood they also saw structures which they interpret as Koch's blue bodies. They conclude that the parasite belongs to the genus *Theileria*, and they name it *T. hirci*. Lestoquard (1924) records as *T. ovis* a parasite of sheep in Algeria (Fig. 430).
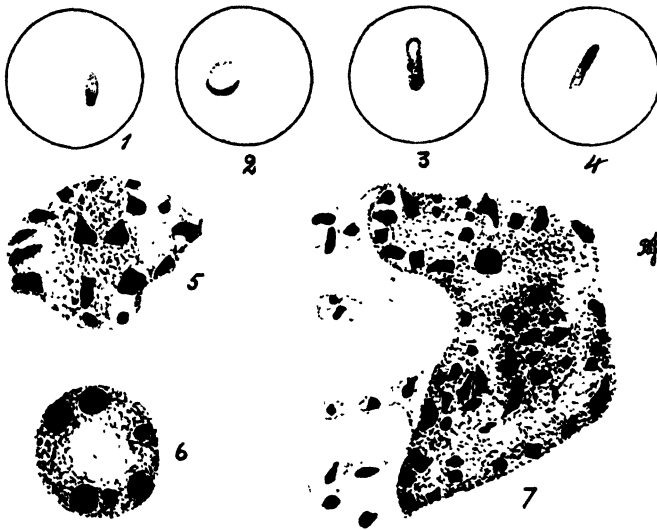
FIG. 430. –*Theileria hirci* OF SHEEP (×ca. 3,000). (AFTER LESTOQUARD, 1924.)

1-4. Forms in peripheral blood.
5-7. Schizonts (blue bodies) from smears of liver and spleen.

Schizonts were found in the liver, spleen, kidneys, and bone marrow. As both goats and sheep are inoculable with the parasite, it seems probable that the form observed by Dschunkowsky and Urodschevich (1924) in goats was the same. The name *T. hirci* may therefore be accepted as the correct name for the *Theileria* of sheep and goats.

According to Lestoquard (1924), the disease is characterized by constitutional disturbances accompanied by fever. On the second day of the disease there may be a transient hæmoglobinuria. A fall in temperature followed by death then occurs. After death all the organs show hæmorrhagic lesions similar to those in cattle which succumb to East Coast fever.

The parasites as they occur in the red blood-corpuscles are very similar to *B. sergenti*, *B. mutans*, and the blood forms of *T. parva*. About 2 per cent. of the corpuscles of the peripheral blood are infected. In blood taken from the spleen the percentage is higher. Schizonts occur in the liver, spleen, kidneys, and lymphatic glands.

Priestly (1915) described as *T. tachyglossi* a small parasite of the red blood-corpuscles of *Tachyglossus aculeatus*, an echidna of Australia. The blood forms resembled those seen in *T. parva* infections of cattle, while in smears of the organs and also in the blood structures resembling the schizonts of the same parasite were said to occur.

The structures described as *T. tsutsugamushi* which Hayashi (1920) recorded from the lesions of tsutsugamushi disease of man in Japan may not be parasites at all.

Action of Drugs on Babesia and Theileria.

Though many drugs, including the various arsenic derivatives, have been employed in the treatment of piroplasmosis, the only drug which has anything like a specific action is trypan blue. As was first shown by Nuttall and Hadwen (1909), a dose of 5 to 10 c.c. of a 1 or 1·5 per cent. solution injected intravenously has the effect of causing *B. canis* in the blood of dogs to degenerate and eventually disappear. In this manner the serious symptoms resulting from heavy infections may be averted. Though the animals recover clinically, they are in the condition of naturally recovered animals, for the blood remains infective for many years. Trypan blue has a similar effect on *B. bigemina*, *B. caballi*, and *B. motasi*, and the drug is recognized as a curative agent. On the other piroplasmata of cattle and horses and the parasite of East Coast fever, *T. parva*, it has no action whatever. It thus appears that trypan blue is specific for the largest of the piroplasmata and not for the smaller forms. This fact has been put forward as an argument in favour of the separation of the large parasites in a separate genus. The distinction is really a physiological one, and, like the immunity reaction, cannot be employed for the separation of genera and species.

Supposed Theileria of Man.

In this connection may be mentioned a supposed *Theileria* which has been described as the cause of tsutsugamushi disease, an infectious condition which occurs along the rivers in the northern provinces of Japan. Kitashima and Miyajima (1918) have given a complete account of the disease. It is produced by the bite of a mite (*Leptus akamushi*), and is characterized by fever which occurs about eight days after exposure to infection. Recovery in about a month is the rule, but death may occur in about ten days. Hayashi (1920) claims to have discovered an organism which he believes is the causative agent, and has elaborated for it a complicated

life-cycle. It occurs in the form of short rods, spheres, or rings, and is found chiefly in endothelial cells at the region of the wound inflicted by the mite, in the lymphatic glands, and the spleen. It may occur free in the plasma and in severe cases in the red cells. In films stained by Romanowsky stains the supposed parasite is seen to consist of blue cytoplasm and red chromatin. It is assumed that reproduction takes place by binary fission as also by schizogony, like that occurring in *T. parva*. Furthermore, Hayashi describes a most complicated life-cycle in which asexual and sexual stages occur. It is impossible to accept these statements till more evidence of the parasitic nature of the structures is forthcoming. Hayashi concluded that he was dealing with a species of *Theileria*, which he named *T. tsutsugamushi*, but the structures may not be parasites at all, though they bear some resemblance to *Rickettsia*. Faust (1923a) believes that the disease may occur in the Yangtse Valley in China, for in smears of the blood and spleen of cases which appear to be similar to those of Japan he has found within red blood-corpuscles and mononuclear cells small bodies which correspond with those described by Hayashi. These intracorpuscular bodies may possibly be *Grahamella* (p. 1056).

UNPIGMENTED PARASITES OF THE RED CELLS OF COLD-BLOODED ANIMALS.

Certain unpigmented parasites of the red blood-corpuscles of reptiles and amphibia have been described. They cannot be included with the hæmogregarines, and it is possible they are the representatives in cold-blooded animals of the mammalian piroplasmata.

Dactylosoma ranarum (Kruse, 1890).—In the blood of frogs there sometimes occurs within the red cells a small parasite which is devoid of pigment, and which multiplies by producing four to sixteen merozoites (Fig. 431). According to Nöller (1913b), who is the most recent observer to study this organism, it was first seen by Kruse (1890) in European frogs. He gave it Lankester's name, *Drepanidium ranarum*. Celli and Sanfelice (1891) wrote of it as *Hæmogregarina ranarum*, and Grassi and Feletti (1892) as *Laverania ranarum*, as they recognized its resemblance to the human malarial parasites. Labbé (1894) studied the organism and placed it in a new genus as *Dactylosoma splendens*. As Kruse (1890) had used the specific name *ranarum*, the parasite becomes *Dactylosoma ranarum* Kruse, 1890. Owing to the fact that gametocytes of an elongate form occur in the red cells, these have been described by various observers as minute hæmogregarines belonging to the genus *Lankesterella*, and much confusion has resulted.

D. ranarum has been seen frequently in European frogs by the above-mentioned observers, and by Ziemann (1898), Hintze (1901), Sambon and Low (1901), and França (1908c). Durham (1902) described the organism from Brazilian toads, and Billet (1904) in Tunis attempted to trace a connection between it and *Trypanosoma inopinatum*. Dutton, Todd, and Tobey (1907) observed the organism in *Rana galemensis* of the

Gambia, while Finkelstein (1908) recorded it from Caucasian frogs. Mathis and Leger, M. (1911a), again met with it in *R. güntheri* of Tonkin. It is evident, therefore, that it has a wide distribution.

According to Nöller (1913b), the asexual stage of the parasite may be seen within the living red cells as hyaline masses of cytoplasm, either elongate or rounded, and containing characteristic globules of a refringent material (Fig. 431). The schizonts have a diameter of 4 to 9 microns, while the number of merozoites produced is four to sixteen. In films stained by Romanowsky stains the parasite consists of blue cytoplasm

FIG. 431.—*Dactylosoma ranarum* FROM THE BLOOD OF FROGS. (1-5 AFTER MATHIS
AND LEGER, 1911; 6-14 AFTER NÖLLER, 1913.)

1-5. Forms in red blood-corpuscles of *Rana güntheri* of Tonkin. Dried films. ($\times$ 1,100.)
6-14. Form in red blood-corpuscles of *Rana esculenta* of Europe. The red cells are not repre-
sented. Wet fixed films. ($\times$ 2,700.)
6-10. Stages in schizogony.   11-12. Microgametocytes.   13-14. Macrogametocytes.

containing a varying number of nuclei, and also other granules which take a red colour. When nuclear multiplication is complete merozoite formation commences, and this takes place frequently by the development of buds on one side of the parasite only, producing eventually a fan-like appearance very similar to that occurring in *Babesia quadrigemina* or *Plasmodium minasense* (Plate XVII., 6-15, p. 982, and Fig. 426). When the number of merozoites produced is only four, the resemblance is very striking. The individual merozoites are slightly elongate bodies measuring 2 to 3 microns by 1 to 1·5 microns. The nuclei are roughly spherical.

Nöller describes a second type of schizogony which gives rise to merozoites having slightly elongate or even dumb-bell-shaped nuclei. This type of merozoite, after entering another red cell, grows into a gametocyte instead of into a schizont (Fig. 431, 11-14). The gametocytes, when fully formed, are about 5 to 8 microns in length by 1·5 to 3 microns in breadth. They usually lie at one end of the red cell, and the narrower end is often bent into a loop. In some of the gametocytes the nucleus, which is a spherical body, contains a small karyosome, while in others there is a much larger one. The former may represent male gametocytes, and the latter female. Nöller was unable to obtain any transmission by the leech, *Hemiclepsis marginata*, and expresses the opinion, though on what grounds is not clear, that the " fish louse," *Argulus foliaceus*, is the probable vector. Owing to the fact already noted that the gametocytes of *Dactylosoma ranarum* are elongate, vermicular structures, it is possible that some of the small parasites which have been supposed to be hæmo-gregarines really belong to this genus. Thus the small one described by Fantham (1905) as *Lankesterella tritonis* may be a *Dactylosoma* of the newt. Awerinzew (1914) described as *Lankesterella amania* a parasite of *Chamæleon fischeri* of West Africa. It occurred as minute spherical bodies in the red cells. One to six nuclei were present, and production of six merozoites was described. The parasite is evidently allied to that of the frog, and should be known as *Dactylosoma amaniæ*.

PARASITES OF DOUBTFUL NATURE.

There occur in either the blood or organs of animals certain parasites which cannot be placed in any natural scheme of classification of the Protozoa. It is possible that in some cases they are not Protozoa at all. The most important of these are the organisms included in the genus *Toxoplasma*. Other forms are *Elleipsisoma*, *Pirhæmocyton*, and *Cytamœba*.

Toxoplasma NICOLLE AND MANCEAUX, 1909.

The organisms included in the genus *Toxoplasma* occur as parasites in the body fluids, endothelial cells, or leucocytes of various vertebrates. They have the form of small elongate, slightly curved masses of cytoplasm with a central nucleus, and are found in the cells either singly or in groups which result from a process of repeated binary fission. When they occur singly they not infrequently lie against the nucleus, which may be indented, so that they bear some resemblance to the leucocytozoa or leucocytic hæmogregarines, to which some of the forms described in birds have been referred. The occurrence of reproduction by binary fission does not

harmonize with this view.   As suggested by Nöller (1920), some of them
may be merozoites of hæmogregarines or coccidia.   Aragão described
parasites from seven South American birds.   He regarded them as hæmo-
gregarines, but Nöller (1920) came to the conclusion that they were
probably toxoplasmata.   Hoare (1924a), who has described a typical
leucocytic hæmogregarine (*Hepatozoon adiei*) from an Indian eagle, has
examined Aragão's description, and finds that in all probability five of
the birds actually harboured hæmogregarines (see p. 1095), but that in
two (*Sporophila albigularis* and *Sicalis flaveola*) the parasites were toxo-
plasmata.   Mayer, according to Nöller (1920), discovered a parasite
which appeared to be a toxoplasma in the spleen and liver of a bird
(*Chrysomitris spinus* L.) in Hamburg.   The bird also had the small
intestine infected with an organism which resembled an *Eimeria*.   There
were male and female gametocytes of the *Eimeria* type and also schizonts.
The latter occurred in the subepithelial tissues, amongst which numerous
merozoites were dispersed.   They were traced in the lymphatics to the
liver, and it appears not improbable that the forms in the liver and spleen
were in reality merozoites of the intestinal organism.   Though some of
the toxoplasmata may be merozoites of hæmogregarines or coccidia, this
cannot apply to such an organism as *Toxoplasma gondii*, which is readily
inoculable from animal to animal, and which multiplies by binary fission.
Till more is known of the life-history and the natural mode of infection
of toxoplasmata it will be impossible to determine their relationship to
other Protozoa.

The first of these organisms to be discovered was *T. gondii* (Nicolle
and Manceaux, 1908), which was found by them (1908) in the gondi
(*Ctenodactylus gundi*) of North Africa (Fig. 432).   In the same year
Splendore described a form (*T. cuniculi* Splendore, 1910) from the rabbit
in Brazil.   Both these parasites are readily inoculable to pigeons.   It
seems probable that a parasite seen by Laveran (1900) in the Java sparrow
(*Munia oryzivora*) was really a toxoplasma.   Adie (1908) described what
was undoubtedly one of these organisms from the common sparrow of
India, and Novy and McNeal (1904) another as *Hæmoproteus rouxei* of the
American sparrow.   De Mello (1910) discovered a dog naturally infected in
Turin, an observation repeated by Yakimoff and Kohl-Yakimoff (1911b)
in Germany, and by Boëz (1921) in Strasburg.   Prowazek (1910) men-
tioned as *T. talpæ* a form seen by him in a Japanese mole.   Carini (1911a)
described a natural infection of a dog and pigeon in Brazil with a form which
he concluded was *T. cuniculi*, since the rabbit parasite originally discovered
by Splendore was readily inoculable to dogs and pigeons.   Bourret (1911)
found a rabbit infected in Senegal, while Brug, den Heyer and Haga
(1925) described an epidemic amongst these animals in the Dutch East

Indies. Sangiorgi (1913) described in Italy *T. musculi* from the mouse (*Mus musculus*) and (1914*a*) *T. ratti* from the rat (*Rattus rattus*), while Carini and Migliano (1916) in Brazil gave the name *T. caviæ* to a form occuring naturally in the guinea-pig. Marullaz (1913) rediscovered the parasite originally seen by Laveran in the Java sparrow, and came to the conclusion that it belonged to the genus *Toxoplasma*, to which he also ascribed the various parasites described as hæmogregarines of birds by Aragão (1911) in Brazil. Marullaz proposed to name it *T. avium*, but Aragão had already written of the parasite as *Hæmogregarina paddæ*, so that the form in the Java sparrow is *T. paddæ*. The parasite described by Todd and Wolbach (1912) as *Leucocytogregarina neophrontis* of the Gambian vulture (*Neophron monachus*) is also a toxoplasma. Laveran and Marullaz (1914) described *T. liothricis* from the Japanese bird, *Liothrix luteus*, and de Mello (1915) as *H. francæ* a toxoplasma of the Indian pigeon. Plimmer (1916*a*) described parasites of this type in a small carnivorous mammal of Madagascar (*Cryptoprocta ferox*) and two birds (*Carpophaga concinna* of the Aru Isles and *Pratincola caprata* of India). He also described a form in a snake (*Coluber melanoleucus*) of Mexico. All these animals examined by Plimmer had died in the Zoological Gardens in London. Walzberg (1923) describes toxoplasmosis of the greenfinch in Germany. Castellani (1913) discovered certain bodies in spleen smears of a man in India who had died of an irregular type of fever associated with enlargement of the spleen. He considered the organism to be a toxoplasma, and gave it the name *T. pyrogenes*. Fedorovitch (1916) claimed to have discovered the same organism in South Russia.

Toxoplasma gondii (Nicolle and Manceaux, 1908).—As already remarked, this organism was discovered in North Africa in the little rodent, *Ctenodactylus gundi*, by Nicolle and Manceaux (1908). It was first thought to be a species of *Leishmania*, but further study convinced its discoverers that it was a totally different organism, and in 1909 they founded the new genus *Toxoplasma* for its reception. Curiously enough, the organism has only been found in animals which have been kept for some time at the Institut Pasteur of Tunis. Newly caught animals have not been found infected. Of 400 animals examined within a month of capture, only two were infected, while of seventy-one examined after a month of confinement, thirty-three were infected. This fact seems to suggest that the infection was contracted at the Institute. In 1916 a dog which had been kept near the infected rodents was also found to be suffering from toxoplasmosis.

Morphology.—The organism is elongate and often has a curved or crescent form (Fig. 432). It measures from 4 to 6 microns in length by 2 to 3 microns in breadth. It is pointed at each end, but frequently one

FIG. 432.—*Toxoplasma gondii* (× 1,750). (AFTER CHATTON AND BLANC, 1917.)

[*For description see opposite page.*

end is more so than the other. The nucleus, which in dried films stained by Romanowsky stain consists of an aggregation of red granules, and in wet fixed films is seen to be spherical and to possess a central karyosome, is slightly nearer the blunter extremity than the other. Towards the other extremity is another structure called by Nicolle and Manceaux the paranuclear body. In Romanowsky films stained after osmic fixation it has the form of a bluish-violet homogeneous body, while in wet fixed films stained by the iron-hæmotoxylin method it appears as a vacuole containing granules (Fig. 432, 14). Examined fresh, the parasites appear quite motionless, though not rigid. They can be seen typically free in the peritoneal exudate of inoculated animals. Nicolle and Manceaux, who first regarded the parasites as related to the leish-mania, attempted to obtain evidence by culture methods of the existence of a flagellate stage. No such form could be found, in spite of the fact that Splendore, who had discovered a toxoplasma of the rabbit in Brazil, claimed to have seen them in dried smears of organs. There seems to be little doubt that Splendore was misled by artifacts. In the peritoneal cavity of animals inoculated intraperitoneally the parasites occur either free in the rather thick gelatinous exudate or within cells, which are chiefly of the mononuclear type. Within the cells they lie in vacuoles in the cytoplasm. Only a single one may be present, or the cytoplasm may be packed with them, either in separate vacuoles or as a single group in a large vacuole. Reproduction is by longitudinal division (Fig. 432, 11). The nucleus first divides by elongation and constriction of the karyosome, and this is followed by division of the cytoplasm. In addition, reproduction by schizogony has been described. The process was first noted by Splendore in the case of *T. cuniculi*. It appears that the so-called schizonts can be explained as masses of organisms, the outlines of which have been obliterated by degeneration or faulty technique. Similar errors have given rise to the idea that schizogony occurs in the case of leishmania, and even trypanosomes (*T. cruzi*). In the case of *T. gondii*, however, Nicolle and Manceaux maintain that schizogony does actually occur (Fig. 432, 6-10). In this process the single parasite within the cell grows into a spherical

1. Various types of spore.
2. Spore with drawn-out extremity.
3. Single spore and one just divided.
4. Spore with dividing nucleus.
5. Two young schizonts.
6. Larger schizont with nuclei dividing.
7. Small schizont having produced a group of spores.
8. Large schizont in cytoplasm of large cell.
9. Group of spores resulting from schizogony.
10. Group of schizonts in smear of lung.
11. Group of spores dividing by binary fission.
12. Group of spores in the cytoplasm of a cell resulting from repeated binary fission.
13. Abnormally-shaped spores.
14. Character of spores when fixed and stained without drying.
15. Merozoites of coccidium from intestine of cat for comparison.

body up to 20 microns in diameter. During growth, repeated nuclear divisions take place till as many as twenty to thirty nuclei are present. Finally, segmentation into a number of organisms occurs. It appears, however, that the more usual method of multiplication, as pointed out by Chatton and Blanc (1917), is by binary fission, and this may take place either extra- or intra-cellularly. So far no observer has been successful in obtaining cultures of the parasite. If this could be done, it might throw light on the affinities of this group of organisms.

Susceptibility of Animals.— Intraperitoneal injection of gondis with infected material from other animals produces an acute infection, leading to death in seven to thirteen days. There is accumulation of exudate in the peritoneal, pleural, and pericardial cavities, and numerous parasites are found in the exudate either free or intracellularly. The spleen and liver show little enlargement in these acute infections, though small numbers of parasites occur here, and also in the blood-stream. The natural infection of gondis is of a more chronic nature, and the animals survive several months. In these cases there is not the extensive exudate formation in the serous cavities as after intraperitoneal injections. The liver and spleen are hypertrophied, and the latter organ contains large numbers of parasites. There is congestion and hepatization of the lungs, and parasites are found here also. In the blood and other organs the parasites are scanty. A large number of other animals have been proved to be susceptible to *T. gondii* after intraperitoneal, intravenous, or subcutaneous inoculation. Dogs, cats, mice, jerboas, guinea-pigs, rabbits, pigeons, Java sparrows, and other small animals have been shown to be susceptible. Rats, on the other hand, have not been infected. The virus injected intraperitoneally or intravenously produces, as a rule, an acute infection, leading to death in one to three weeks, according to the size of the animal. Subcutaneous injection is less certain to produce infection, which, if it occurs, is of a more chronic character. It is a remarkable fact that the organism is inoculable into such a variety of hosts, and in this respect it differs from other pathogenic Protozoa which show a much greater specificity. The fact that the virus from the gondi will infect rabbits, guinea-pigs, dogs, mice, moles, pigeons, and Java sparrows, in all of which naturally occurring toxoplasma infections have been found, raises the question of the identity of these various forms.

Brug, den Heyer and Haga (1925) observed an epidemic amongst rabbits in the Dutch East Indies which apperaed to be due to infection with a *Toxoplasma*. *Post-mortem* the spleen was enlarged and dotted over with necrotic areas, which also occurred in the liver and lungs. Parasites were discovered in the spleen, liver, lungs, and heart blood. Numerous multinucleated bodies from 4·5 to 38 microns in diameter occurred. These

were regarded as schizonts, which were destined to break up into spores. In addition, many small bodies with two nuclei were seen, as also the small uninucleated curved spores, which were frequently grouped in masses in spaces in the tissues. The resemblance of certain stages to the schizonts of coccidia and of others to *Encephalitozoon cuniculi* is noted (Figs. 322, 323).

Other Species of Toxoplasma.

As will be seen from the list given below, numerous species have been described. In most cases these have only been studied in smears of the organs of animals which have been killed or died. In some cases it is even doubtful if the structures described as *Toxoplasma* are actually of this nature. It has been already remarked that in the same year that Nicolle and Manceaux discovered *T. gondii*, Splendore in Brazil discovered *T. cuniculi* in the rabbit. With this parasite, which is morphologically indistinguishable from that of the gondi, Splendore (1909) was able to infect rats, guinea-pigs, rabbits, and frogs, while Carini (1909) infected pigeons. With the naturally occurring dog parasite, *T. canis* of Brazil, Carini (1911a) infected rabbits, and Carini and Maciel (1913) dogs and pigeons. Carini and Migliano (1916) discovered *T. caviæ* in guinea-pigs, and showed that it was inoculable to pigeons. It will thus be seen that there are no means of distinguishing the various named species.

Recorded Species of Toxoplasma.

Man: *T. pyrogenes*, Castellani, 1913, Ceylon; Fedorovitch, 1916, Black Sea.

Monkey (*Mycetes seniculus*) : *T.* sp. Theze, 1916, Guiana.

Dog (*Canis familiaris*) : *T. canis*, de Mello, 1910, Italy; Carini, 1911, Brazil; Yakimoff, 1911, Germany; Carini and Maciel, 1913, Brazil; Fedorovitch, 1916, Black Sea; Blanc, 1917, Tunis; Boëz, 1921, Strasburg.

Fossa (*Cryptoprocta ferox*) : *T.* sp. Plimmer, 1915 (Zoological Gardens, London), Madagascar.

Gondi (*Ctenodactylus gundi*) : *T. gondii*, Nicolle and Manceaux, 1909, Tunis.

Rabbit (*Lepus cuniculus*) : *T. cuniculi*, Splendore, 1908, Brazil; Bourret, 1911, Senegal.

Rat (*Rattus rattus*) : *T. ratti*, Sangiorgi, 1914, Italy; v. Saceghem, 1916, Congo.

Mouse (*Mus musculus*) : *T. musculi*, Sangiorgi, 1913, Italy.

Guinea-pig (*Cavia cobaya*) : *T. caviæ*, Carini and Migliano, 1916, Brazil.

Squirrel (*Sciurus* sp.): *T. sciuri*, Colos, 1914, England.

Mole (*Talpa* sp.): *T. talpæ*, Prowazek, 1910, Japan.

Birds:

> Columba domestica : *T. columbæ*, Carini, 1911, Brazil; *T. francæ*, de Mello, 1915, India.
>
> Sporophila albigularis : *T. sporophilæ*, Aragão, 1911, Brazil.
>
> Sicalis flaveola : *T. sicalidis*, Aragão, 1911, Brazil.
>
> Neophron monachus : *T. neophrontis*, Todd and Wolbach, 1912, Gambia.
>
> Munia orizivora : *T. paddæ*, Aragão, 1911; *T. avium*, Marullaz, 1913; Laveran 1900 (Java sparrow, locality ?).

Birds : *continued—*

  Liothrix luteus : *T. liothricis,* Laveran and Marullaz, 1914, Japan.

  Carpophaga concinna : *T.* sp. Plimmer, 1915 (Zoological Gardens, London), Aru Isles.

  Pratincola caprata : *T.* sp. Plimmer, 1915 (Zoological Gardens, London), India.

  Turdus rufiventris : *T.* sp. Carini and Maciel, 1916, Brazil.

  Volatinia jacarini : *T.* sp. Carini and Maciel, 1916, Brazil.

  Aaptus chopi : *T.* sp. Carini and Maciel, 1916, Brazil.

  Pitangus sulphuratus : *T.* sp. Carini and Maciel, 1916, Brazil.

  Elœnca albiceps : *T.* sp. Carini and Maciel, 1916, Brazil.

  Gypagus papa : *T.* sp. Carini and Maciel, 1916, Brazil.

  Sparrow: *T.* sp. Adie, 1908, India.

Snake (*Coluber melanoleucus*) : *T.* sp. Plimmer, 1916 (Zoological Gardens, London). Mexico.

Supposed Toxoplasma of Man.

Toxoplasma pyrogenes Castellani, 1914.—Under this name Castellani (1914*b*) described certain structures which he found in smears of the blood and spleen of a man who died in Ceylon. Their probable nature was discussed by the writer (1923). They are round, ovoid, or piriform in shape, and about 6 microns in diameter (Fig. 433, A-C). A form 12 microns in diameter and containing several chromatin granules is described as a schizont. From Castellani's diagrammatic figures (Fig. 443, B) it is impossible to judge of their nature, but Plate (1914), who saw the films, came to the conclusion that they represented some unknown Protozoon which it was impossible to classify. He gives two figures which appear to have been carefully drawn (Fig. 433, A). These resemble vegetable cells like yeasts more than any other organism, and it is evident Plate did not realize the possibility of tissues becoming contaminated after death. There is no evidence that the bodies are Protozoa at all, much less toxoplasmata. They are almost certainly contaminating organisms of a vegetable nature. The structures seen in the blood of a child by Fedorovitch (1916) on the Black Sea coast (Fig. 433, C) and which he regarded as nearly related to *T. pyrogenes,* are without doubt cocci, large bacilli, or yeasts which had contaminated the films. Chalmers and Kamar (1920) claimed to have discovered *T. pyrogenes* in a case of splenomegaly in the Sudan, but, as pointed out by the writer (1922), they were dealing with altered leishmania. It is perfectly clear that no such parasite as *T. pyrogenes* exists. It should always be remembered that vegetable cells, when stained in dried films with Romanowsky stain, take the characteristic red and blue colours (Fig. 433). Such organisms may find their way into the spleen or other organs from the intestine after death, they may appear in smears because the slides were contaminated before the films were made, or they may fall on the film from

A. *Toxoplasma pyrogenes* as depicted by Plate (1914) from films made by Castellani.

B. *Toxoplasma pyrogenes* as depicted by Castellani (1914). 1-4, Small forms; 5, large form, described as schizont.

C. *Toxoplasma pyrogenes* as depicted by Fedorovitch (1916).

D. Supposed hæmogregarine of trench fever as depicted by Dimond (1917).

E. Vegetable cells depicted by Balfour (1914) from a contaminated blood-film of a Sudan cob. The form E 3 bears a striking resemblance to a hæmogregarine.

F. Vegetable cells and other organisms described by Elders (1911) as Protozoa in human blood. The form at F 3 may be a *Euglena* or *Astasia.*

G. Vegetable cells described by Manson (1905) as possibly Protozoa from a spleen smear made *post-mortem* from a case of kala-azar.

H. Vegetable cells described by Castellani and Willey (1905) from human blood. Castellani and Chalmers (1919) make the suggestion that they may be spores of *Sarcocystis.*

I. Vegetable cells seen in spleen smear of a case of kala-azar by Bousfield (1911).
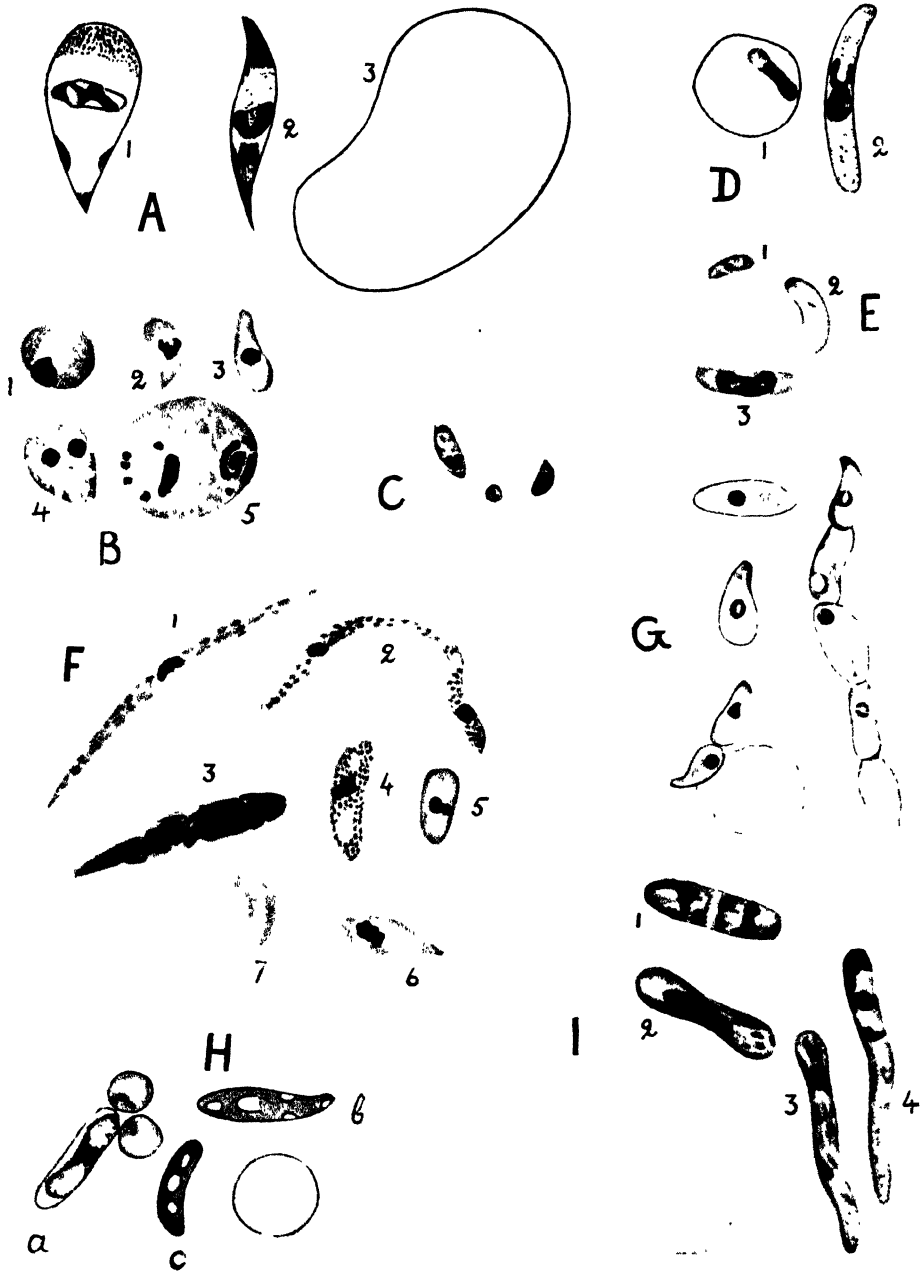
FIG. 433.—VARIOUS BODIES, PROBABLY OF VEGETABLE NATURE, WHICH HAVE BEEN ERRONEOUSLY DESCRIBED AS PARASITIC PROTOZOA. (AFTER WENYON, 1923, FROM *Tropical Diseases Bulletin*, vol. xx., p. 545.)

The magnification is indicated by the red blood-corpuscles.

the air. At other times they occur in the distilled water used for staining, and are deposited on the film. Rocha-Lima (1912) drew attention to the fact that yeasts stained in this manner frequently simulated Protozoa, and pointed out that *Histoplasma capsulatum* and *Cryptococcus farcinimosus*, both of which had been regarded as Protozoa, were in reality yeast-like organisms (Plate III., 1-2, p. 394). Fedorovitch's observation, noted above, is quoted by Castellani and Chalmers (1919) in support of the specific nature of this parasite. The figures given by Fedorovitch show structures which are unquestionably contaminating organisms.

FIG. 434. — *Elleipsisoma thomsoni*
FROM THE BLOOD OF THE MOLE
(× 1,200). (AFTER J. D. THOMSON,
1906.)

1-4 and 6. Intracorpuscular forms   The
        cell is considerably enlarged
5. Form free in plasma.

Elleipsisoma FRANÇA, 1912.

The curious parasite which was named *Elleipsisoma thomsoni* by França (1912*b*) was first discovered by Thomson, J. D. (1906), in moles in England. It was subsequently studied by França (1911*a*) in Portugal. It resembles a *Babesia* in being devoid of pigment, but differs in other respects. It appears to be either within the red cells or free in the plasma (Fig. 434). The largest forms may reach a diameter of 8 microns. Within the cells the adult parasites are usually ovoid, and measure from 6 to 7·5 microns in length by 4·5 to 6 microns in breadth. Multiplication forms were seen only in smears of the lungs, and the process takes place by binary fission, whereby two somewhat elongate individuals are produced, and possibly by a multiple division, as forms with as many as five chromatin masses were seen. The figures given by França of the division forms are not very convincing, and it is evident that the parasite requires further investigation. Because it differed in certain respects from the piroplasmata, França placed it in a new genus as *E. thomsoni*.
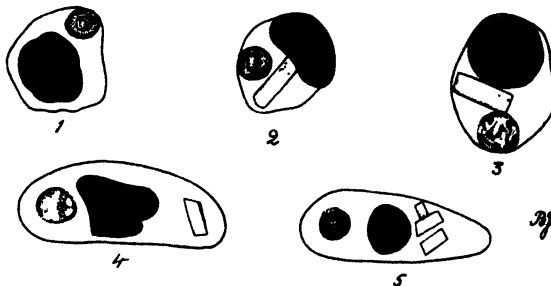
FIG. 435.— *Toddia bufonis* FROM BLOOD OF THE TOAD, *Bufo regularis*
(× *ca.* 1,500). (AFTER FRANÇA, 1910.)

In addition to the parasite the red cell contains one or more crystalline rods.

Cytamœba LABBÉ, 1894.

Dutton, Todd and Tobey (1907) described certain intracorpuscular bodies which they had seen in the blood of frogs in the Congo. They occurred as red staining spherical structures, which were either homogeneous or granular. Asso-

ciated with them were rods of a crystalline nature. They had an average diameter of about 2 microns, and were referred to as cytamœbæ. Mathis and Leger (1911a) describe them from *Bufo melanostictus* of Tonkin. Whether they are actually parasites or not it is difficult to state, but França (1910), who encountered them in *Bufo regularis* of Portuguese Guinea, regarding them as such, established the genus *Toddia* for their reception, and named the form seen by him *Toddia bufonis* (Fig. 435). More recently, Hegner (1921a) has given a description of the structures described by Labbé (1894) as *Cytamœba bacterifera* from the blood of European frogs. Hegner discovered the bodies in the red cells of *Rana clamitans* and *R. catesbiana* of North
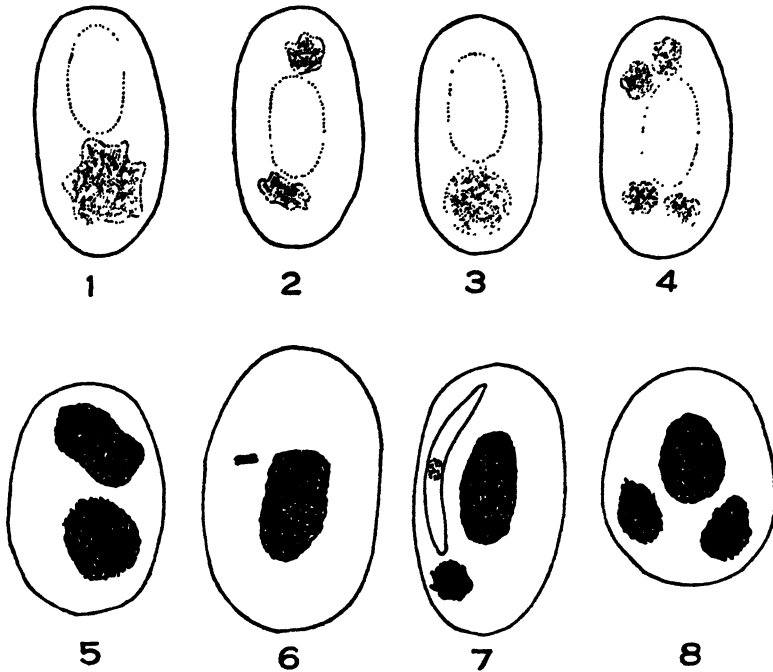
Fig. 436. - *Cytamœba bacterifera* IN THE RED BLOOD-CORPUSCLES OF FROGS (× 1,440).
(AFTER HEGNER, 1921.)

1-4. Red cells containing one to four " cytamœbæ," as seen in fresh blood.
5. Rounded " cytamœba," as seen in stained film. It appears as a mass of bacilliform bodies.
6. Very young form appearing in stained film as two bacilliform bodies.
7. Stained red cell containing a " cytamœba " and a hæmogregarine (*Lankesterella*).
8. Stained red cell with two " cytamœbæ."

America. They occurred as rounded structures from 3 to 7 microns in diameter, and were situated towards the end of the red cell (Fig. 436). When observed in the freshly-drawn blood, they exhibited for a few minutes active amœboid changes of shape, while the bacillus-like structures which occurred within the bodies, and which have been named *Bacillus krusei* by Laveran (1899a), were in active move-ment. The amœboid movements of the bodies quickly ceased, but the rods con-tinued to move about for several hours. The amœboid changes of shape which occur at first incline this observer to regard the bodies as parasites within which a bacillus is living either in symbiosis or as a hyperparasite. In stained specimens,

however, no nucleus could be detected, the rods appearing like red staining bacilli. The view that the bodies are produced by the red blood-corpuscle as a reaction to bacterial invasion is a tempting one, though in this case it would be difficult to account for the amœboid movements.

Pirhæmocyton CHATTON AND BLANC, 1914.

Chatton and Blanc (1914a, 1916) noted within the red cells of the North African gecko, *Tarentola mauritanica*, a peculiar organism which has some resemblance to a piroplasma (Fig. 437, 1-6). The smallest forms resembled anaplasmata, and appeared in stained films as red dots about 1 micron in diameter. These led up to larger circular individuals measuring 3 to 4 microns in diameter, and consisting of clear cytoplasm and a central chromatin dot. In some cases the chromatin dot appeared to be dividing. The presence of the parasite in the red cell was associated with the appearance of a globular albuminous body in another part of the cell.
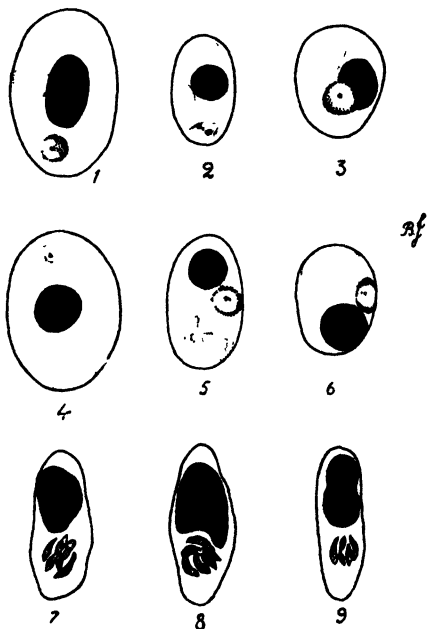
FIG. 437. - PARASITES IN THE RED BLOOD-CORPUSCLES OF THE GECKO, *Tarentola mauritanica*, OF TUNIS (× 1,000). (AFTER CHATTON AND BLANC, 1914-1916.)

1-6. *Pirhæmocyton tarentolæ*. Besides the parasite, the cells contain one or more globular albuminous bodies.   7-9. Unnamed parasite.

This body varied in diameter up to 8 microns, and in the fresh condition appeared to be homogeneous and refractile. The name given to this supposed parasite was *Pirhæmocyton tarentolæ*.

In the same animals another intracorpuscular parasite had been previously described by these authors (1914). It consisted of a tiny, elongate, oval, or sickle-shaped body, groups of five to ten of which occurred within vacuoles (Fig. 437, 7-9). Each possessed two chromatin granules, one larger than the other, producing an appearance of a minute elongate leishmania. Nothing is known of the origin or development of either of these parasites.

INTRACELLULAR STRUCTURES OF DOUBTFUL NATURE.

There has been described under various names a number of intra-cellular bodies, the nature of which has not been satisfactorily determined. Some of them may certainly be parasites, but even this is not clear. The best known of these are *Anaplasma, Grahamella,* and *Rickettsia.* Some hold that *Anaplasma* is a parasite, and others that it is a degenerative change occurring in the red blood-corpuscles as a result of some unknown virus. The structures described under the name *Grahamella* are regarded by some observers as definite parasites, and by others as merely granules resulting from basophilic changes in the red cells. Those grouped under the name *Rickettsia* are possibly organisms related to the bacteria. These various structures will be considered under their respective names.

Anaplasma THEILER, 1910.

In their study of Texas fever, Smith and Kilborne (1893) described certain red granules, which occurred on the margins of the red blood-corpuscles of cattle. They called these granules " marginal points," and concluded that they represented a chronic or resistant phase of *Babesia bige-mina.* Theiler (1910) studied these bodies in South Africa, and came to the conclusion that they represented a distinct parasite, to which he gave the name *Anaplasma marginale.* Since then the bodies have been seen by many observers, and there has been much controversy as to their real nature. They occur very commonly in association with *B. bigemina* and *B. mutans,* but Theiler (1912) in South Africa and Lignières (1919) in the Argentine recorded instances in which cattle have been subject to pure infections of these bodies. It seems probable that cattle all over the world are liable to have them in their blood. Theiler distinguished two forms (Fig. 438), one which showed a tendency to be near the margin of the red cell (*A. marginale*) and the other towards the centre (*A. marginale* var. *centrale*). Theiler stated that animals which were immune to one form were inoculable with the other. From this time onwards the anaplasmata were described by numerous observers in various parts of the world without anything definite being discovered. Some regarded them as parasites, others as granules analogous to those which appear in red cells in anæmic conditions. Within the red cells of man and animals there frequently occur, especially in young animals or in anæmic conditions, red-staining granules, which are generally known as " Jolly bodies." These are spherical structures, which may reach a diameter of 0·5 micron. They are never very numerous in the blood, but morphologically they can hardly be dis-tinguished from the anaplasmata which occur in the blood of cattle.

They are generally supposed to represent the remains of the nucleus of immature red cells, so that the cells containing them may be regarded as nucleated red cells with very minute nuclei. Laveran and Franchini (1914) described them as occurring in numerous animals, including rats, mice, rabbits, guinea-pigs, moles, cats, dogs, calves, pigs, and monkeys. They repeated an experiment first made by Dias and Aragão (1914), who showed that the bodies appeared in animals injected with phenylhydrazine.

Morphology.—The anaplasma body, as it occurs in cattle, is a spherical granule which stains a bright red with Romanowsky stains, and varies

A   **B**
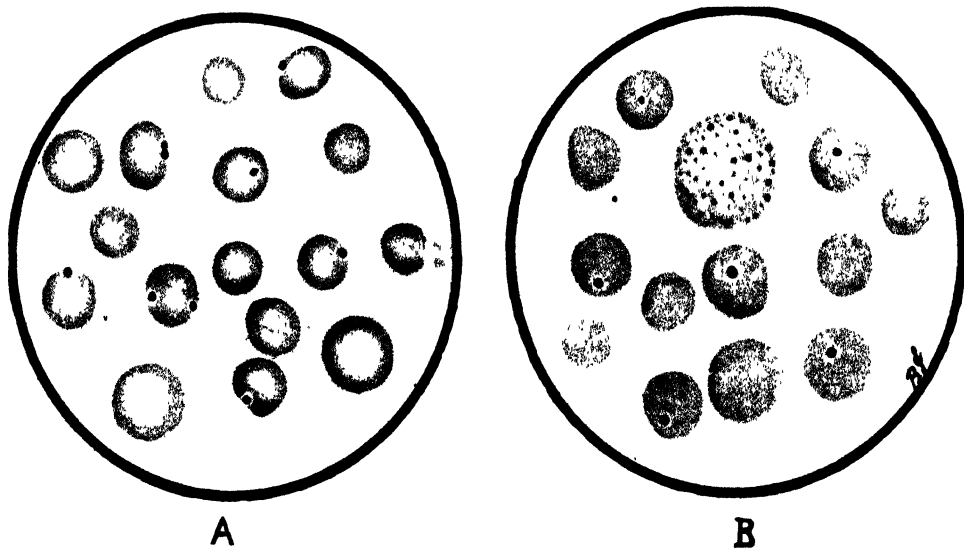
FIG. 438.—ANAPLASMOSIS OF CATTLE (× ca. 2,000). (ORIGINAL.)

A. *Anaplasma marginale.*
B. *Anaplasma centrale;* an enlarged red blood-corpuscle showing basophilic spots is also
shown.

in size from 0·1 to 0·5 micron. It shows no structure, though a halo may sometimes be seen around it. This is probably a purely mechanical effect. The fact that two of these bodies may be in contact, producing a dumb-bell appearance, has suggested a multiplication by division. Theiler supposed the anaplasmata to be Protozoa, consisting entirely of chromatin, but actually there is no evidence that they have a protozoal nature, even if they are to be regarded as parasites at all. In some of the smaller piroplasmata, such as *Babesia mutans*, as seen in dried blood-films, the cytoplasm may be reduced to such an extent that the nucleus alone remains visible. It is this fact, together with their frequent association with piroplasmata,

which has led observers to regard them as protozoal organisms, to no known forms of which do they bear any real resemblance.

Lignières (1919) published accounts of the condition as it occurs in South American cattle, and his is the only recent contribution to the subject which has added to our knowledge of these problematic structures. This observer notes that animals may show "Jolly bodies," which are never inoculable from one animal to another, whereas the anaplasmata not only occur in the blood in large numbers, as many as 50 per cent. of the corpuscles being affected and many of them containing several anaplasmata, but they are readily inoculable to cattle which have not previously been infected.

Furthermore, he has shown that sheep and goats can be inoculated, and in these the infection may be maintained indefinitely by sub-inoculations. Other animals, such as the guinea-pig, rabbit, pig, and horse, are not inoculable. The sheep and goats are not affected by the inoculation, except by a transitory rise in temperature at about the thirtieth day. There is no visible change in the blood, and anaplasmata cannot be found there, but that they are present is proved by the fact that the blood produces typical attacks, with appearance of anaplasmata, when inoculated to susceptible cattle. It is concluded that in the blood of sheep and goats the anaplasma is present in a form too small to be observed. As sheep and goats cannot be infected with *Babesia bigemina* or *B. argentina*, with which the anaplasma is often associated in cattle, inoculation of these animals affords a means of separating pure strains of anaplasma. Furthermore, the infection produced in cattle by inoculation from the goat or sheep is never so severe as that produced directly from cattle, so that the infection in these animals may be employed as a means of vaccinating cattle against infections.

In the ordinary course of events, cattle which become infected suffer from fever and intense and progressive anæmia, the red cells being reduced to a quarter of their number in a few days. The mortality rate varies considerably, but for adult animals it may be as high as 95 per cent., and for young ones 50 per cent. After recovery there is complete immunity to reinfection, but, as in the case of the true piroplasmata, the blood remains infective for many years.

Lignières (1924, 1924a) has succeeded in infecting cattle and sheep in France with a strain brought from South America in sheep. Lestoquard (1924a) has given the name *Anaplasma ovis* to the form which occurs in sheep and goats in Algeria, but in view of Lignières' observation it is doubtful if it is distinct from the form in cattle.

Transmission.—Theiler (1912a) described experiments with *Margaropus decoloratus*. Larvæ which had fed on infected animals produced infection

of *A. marginale* in healthy animals after an incubation of fifty-two to seventy-eight days. In another experiment, larvæ of *Rhipicephalus simus* which had been hatched from eggs laid by adults taken off infected animals produced the infection. In a similar manner, Theiler claimed to have transmitted *A. centrale* by means of *M. decoloratus* which had been hatched from the egg. From these experiments Theiler concluded that the virus is able to pass through the egg. Neither Lignières (1919) nor Brumpt (1920) has been able to confirm Theiler's observations, though they have conducted many experiments.

Helm (1924), working in Germany with a strain of the virus obtained by inoculating local cattle with the blood of animals imported from Texas, states that larvæ of *Ixodes ricinus*, hatched from eggs laid by ticks which had fed on infected cattle, were able to transmit the infection. There was an incubation period of two months.

Anaplasmata have been described from animals other than cattle. Thus Schellhase (1912–1914) has recorded its presence in sheep, goats, and donkeys of Africa; Bevan (1912) in sheep in Rhodesia; Basile (1912) in dogs in Italy; Yakimoff and Schokhor (1917) in cattle, Yakimoff and Koselkien (1917) in horses, and Yakimoff (1917a) in dogs in Turkestan. It is difficult to decide in these cases whether the authors were actually dealing with anaplasmata or "Jolly bodies." Apparently, the only test of the true anaplasma is the fact that large infections appear after inoculation of susceptible animals, and that immunity occurs after recovery.

Though the experiments of Theiler and the later ones of Lignières seem to establish the fact that the presence of the bodies in the blood is associated with a definite infection, they do not prove absolutely that the anaplasmata themselves are parasites. Veglia (1915) claims to have cultivated *A. marginale* in South Africa. He states that multiplication occurs in the cultures by binary or multiple fission. Unfortunately, animals do not appear to have been inoculated from the cultures, so that, again, there is no proof that the forms seen in the cultures were not cocci. As "Jolly bodies," which are hardly recognizable from anaplasma, occur especially in anæmic conditions, it is just possible that some invisible virus produces a disease in cattle, one of the features of which is the production of large numbers of "Jolly bodies." The experiments of Lignières with sheep and goats seem to be in favour of this view, for, though the virus was present in the blood, anaplasma did not appear.

Grahamella BRUMPT, 1911.

Graham-Smith (1905) described what he considered to be a new form of parasite which occurred in the red cells of English moles (Fig. 439). The infected cells were occupied by minute rounded, oval, or rod-like bodies, each of which showed irregular staining, so that a blue cytoplasmic part could be distinguished from a red portion.
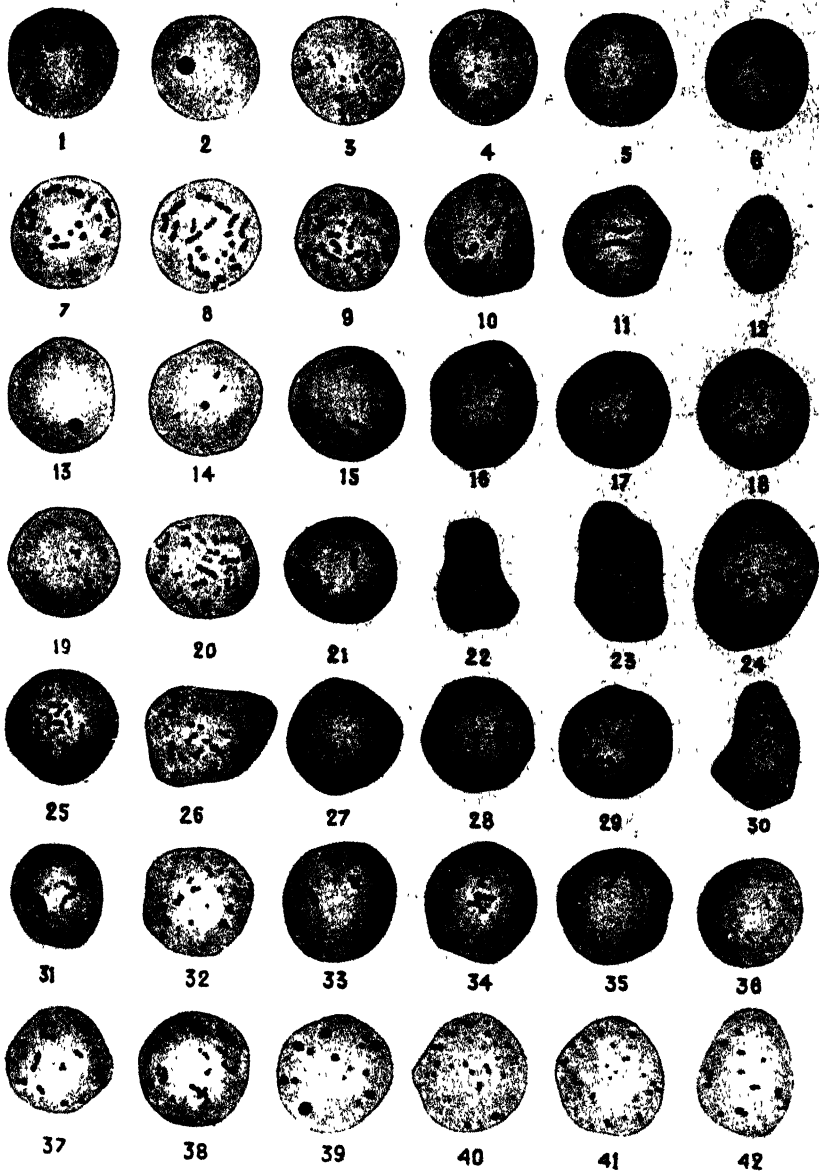
FIG. 439.—*Grahamella* AND BASOPHILIC SPOTS IN THE RED BLOOD-CORPUSCLES OF VARIOUS MAMMALS TO ILLUSTRATE THE VIEW THAT THE TWO ARE IDENTICAL (× *ca.* 2,000).   (AFTER LAVERAN AND MARULLAZ, 1914.)

1-12. Mole.	13-21. Jerboa.	22-24. Field vole.
25-26. Shrew.	27-29. Garden dormouse.	30. Field mouse.
31-35. New-born rat.	36-38. New-born mouse.	39-42. Anæmic cattle.

The rod-shaped or bacillary form is the most characteristic, and this is either straight or slightly curved. Forms with a red granule at each end and a constriction at the middle suggested a reproduction by binary fission. The infected cells might contain only one or two of these bodies, or as many as fifty might be present in a single cell. In some respects the rod forms resemble bacilli showing banded or bipolar staining. These structures have been seen by many observers in small mammals, and there is considerable difference of opinion as to their real nature. Laveran and Marullaz (1914a) came to the conclusion that they represented a change in the red cell analogous to basophilic degeneration which takes place in anæmic conditions (Fig. 439). It was thought that the granules which appear in the red cells in this condition, and which are generally small dots with an irregular or circular outline, may become rod-like and assume the appearance of the bodies under discussion. It was possible to trace gradations from the more irregular granules, which everyone admits to be an indication of basophilic degeneration, to the more uniformly shaped rods of the *Grahamella* type. Graham-Smith, who first described them, and Brumpt regard them as parasitic, and probably of a protozoal nature. Brumpt maintains that the elongate forms multiply by constriction and division at the middle. Brumpt (1911) founded the new genus *Grahamella*, and gave to the form seen in the mole the name of *Grahamella talpæ*. Those occurring in other animals have in some cases been given specific names by Brumpt. Even if the structures prove to be parasites, and at present there is little real evidence of this, it has still to be demonstrated that they are Protozoa. Structurally, they resemble bacilli more than any other organisms, and until more is known of their origin it is safer to regard them as a special type of basophilic granule. Lavier (1921a) draws attention to their marked resemblance to some of the structures described as *Rickettsia*. The following forms have been recorded:

Recorded Species of Grahamella.

Mole (*Talpa europæa*) : *G. talpæ* Brumpt, 1911—Graham Smith (1905), England; Thomson, J. D. (1906), England; Brumpt (1911), France; Visentini (1913), Italy; Coles (1914), England; Laveran and Marullaz (1914), France.

Jerboa (*Jaculus jaculus* and *J. gordoni*) : *G. balfouri* Brumpt, 1911— Balfour (1906), Sudan; Laveran and Marullaz (1914), North Africa.

Vole:
   (*Microtus incertus*) : França (1911), Portugal.
   (*Microtus agrestis*) : Henry (1913), England.
   (*Microtus arvalis*) : *G. microti* Lavier (1921), France.
   (Vole, sp.?): Yakimoff (1917), Transcaucasia.
   (Field vole, sp.?): Coles (1914), England.
   (Water vole, sp.?): Coles (1914), England.

Dormouse (*Myoxus nitela=Elyomys quercinus*) : *G. francai* Brumpt, 1913—França (1911), Portugal; Laveran and Marullaz (1914), France.

Shrew:
   (*Crossopus fodiens=Neomys fodiens*) : Henry (1913), England.
   (Shrew, sp.?): Laveran and Marullaz (1914), France.
   (*Crocidura stampflii*): Leger, A. (1917), West Africa.

Rat:
   (*Rattus rattus*) : Joyeux (1913), French Guinea; Macfie (1917), West Africa
   (*Rattus norvegicus*) : Macfie (1914), West Africa; Carini (1915), Brazil.
   (*Rattus maurus*) : Leger, A. (1913), Upper Senegal.
   (*Golunda fallax*) : *G. joyeuxi* Brumpt, 1913; Joyeux (1913), French Guinea.

Rat—*continued:*

    (*Cricetomys gambianus*) : Macfie (1916, 1917), West Africa.

    (*Acodon serrensis*) : *G. acodoni* Carini (1924), Brazil.

    (Rat, sp.?): Balfour (1911), Sudan.

    (Brown rat, sp.?): Macfie (1914, 1916), West Africa.

    (Field rat, sp.?): Macfie (1917).

    (Young rats, sp.?): Coles (1914), England.

Mouse:

    (Yellow mouse, sp.?): *G. musculi* Benoit-Bazille, 1920, France.

    (*Mus musculus*) : Prowazek (1913), Cameroons.

    (Field mouse, sp.?): Coles (1914), England.

Hamster (*Cricetus phocu*) : *G. ninæ kohl-yakimovi* Yakimoff, 1917, Transcaucasia; Dudstchencko (1914), Transbaikal.

Small rodent (sp.?): Dudstchencko (1914), Transbaikal.

Ox: *G. bovis* Marzinowsky (1917), Russia.

Bat (*Desmodus rufus*) : *G. brumpti* Ribeyro and del Aquila, 1918, Peru. Lavier has informed the writer that he and Larrousse have seen a similar form in *Rhinolophus ferrum-equinum* of France.

Monkey (*Macacus rhesus*) : *G. rhesi* Leger, A., 1922, Annam.
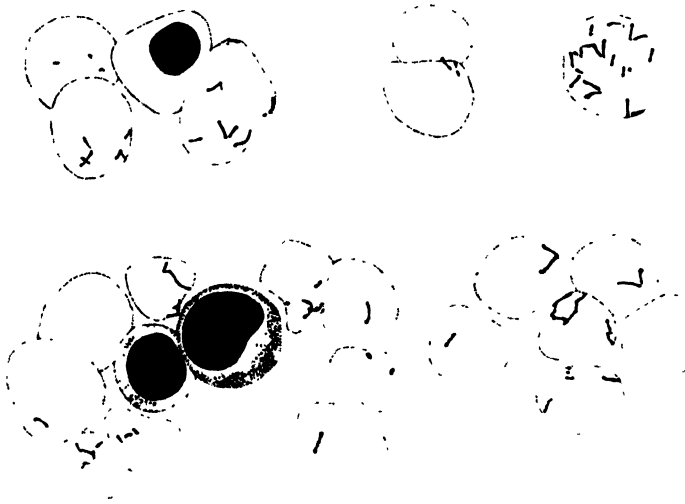
Fig. 440. - *Bartonella bacilliformis* IN THE RED BLOOD-CORPUSCLES OF CASES OF OROYA FEVER (× *ca*. 2,000). (AFTER STRONG, TYZZER, BRUES, SELLARDS, AND GASTIABURU, 1915.)

Bartonella STRONG, TYZZER, BRUES, SELLARDS, AND GASTIABURU, 1915.

In South America, in Peru, there have existed from remote times two diseases known as Oroya fever and Verruga peruviana. The former is characterized by fever associated with marked anæmia, and the latter by a nodular eruption of the skin. For a long time the two diseases were considered as one, but that they are distinct seems to have been established by the American Commission (Strong, Tyzzer, Brues, Sellards, Gastiaburu) in 1913. Oroya fever is of interest in the

present connection, for in association with the marked anæmia there appear in the red cells, and to a less extent in endothelial cells, curious dot and rod-like bodies to which the American Commission gave the name *Bartonella bacilliformis* (Fig. 440). These structures bear some resemblance to *Grahamella* described above. They occur in various forms, the most characteristic of which are slender rods, either straight or slightly curved, and measuring from 1·5 to 2·5 microns in length by 0·2 to 0·5 micron in thickness. Rounded bodies 0·5 to 1 micron in diameter also occur. In dried films stained by Romanowsky stains they appear red, and the rods may show irregular staining and sometimes a slight thickening or knob at each end. An individual cell may contain only a single one, or many may be present, producing a kind of network of fibres through the stroma. In the living conditions, as observed in fresh blood, these rods were noted to change their position in the cell. It is supposed that there is a cycle of development similar to that of *Theileria parva*, and that a multiplication within the endothelial cells leads to the appearance of certain forms which invade the red cells. Attempts to transmit the blood forms to lower animals did not succeed. It seems that evidence of the parasitic nature of the structures in the red cells is far from conclusive.

Mayer (1925) gave the name *Bartonella muris* to similar structures which appeared in the red blood-corpuscles of mice treated with Bayer 205 for trypanosome infections. Mayer, Borchardt and Kirkuth (1926) have shown that the same bodies appear in an anæmic condition following splenectomy in rats. They believe that the operation had stimulated a latent infection.

Rickettsia Rocha-Lima, 1916.

In typhus and Rocky Mountain fever there occur within proliferated endothelial cells of the blood-vessels minute spherical or rod-shaped structures which have been regarded by some observers as organisms of a protozoal nature. Ricketts and Wilder (1910) described small Gram-negative bipolar staining bacilli in the blood of typhus cases. Similar bodies were seen by Hegler and Prowazek (1913) within leucocytes. Wolbach, Todd and Palfrey (1922) were unable to confirm these observations on the blood. Rocha-Lima (1916) discovered that lice which had fed on typhus patients, and which had been proved by Nicolle, Comte and Conseil (1909) to be the vectors of the disease, harboured in large numbers the structures to which he (1916a) gave the name *Rickettsia prowazeki* (Fig. 441, 1-5). Using lice which could not have been naturally infected, Wolbach, Todd and Palfrey (1922) found that the organism occurred only in those lice which were allowed to feed on typhus cases. What are supposed to be the same organisms were found by these observers and others in the intravascular endothelial nodules described by Fraenkel (1914) as pathognomonic of typhus fever. Bodies of a similar nature were seen by Töpfer (1916) in lice fed on cases of trench fever. To these he gave the name of *R. quintana*. Munk and Rocha-Lima (1917) again saw the organism in lice fed on trench fever cases, and also in apparently healthy lice. To those occurring in the latter the name *R. pediculi* was given (Fig. 441, 6). Another form was described by Töpfer (1917) in lice fed on cases of " trench nephritis." *R. prowazeki* is found in lice almost entirely in the gut cells, while the other forms are a little larger and occur in the lumen of the intestine.

Similar structures have been demonstrated in the case of spotted fever of the Rocky Mountains, the virus of which occurs in various rodents, and is conveyed to man by the tick *Dermacentor venustus*. King (1906) and Ricketts (1906) were the first to demonstrate transmission by this tick, while Parker (1923) has incriminated the tick *Hæmaphysalis leporis* as a vector amongst rabbits in nature. Wilson and
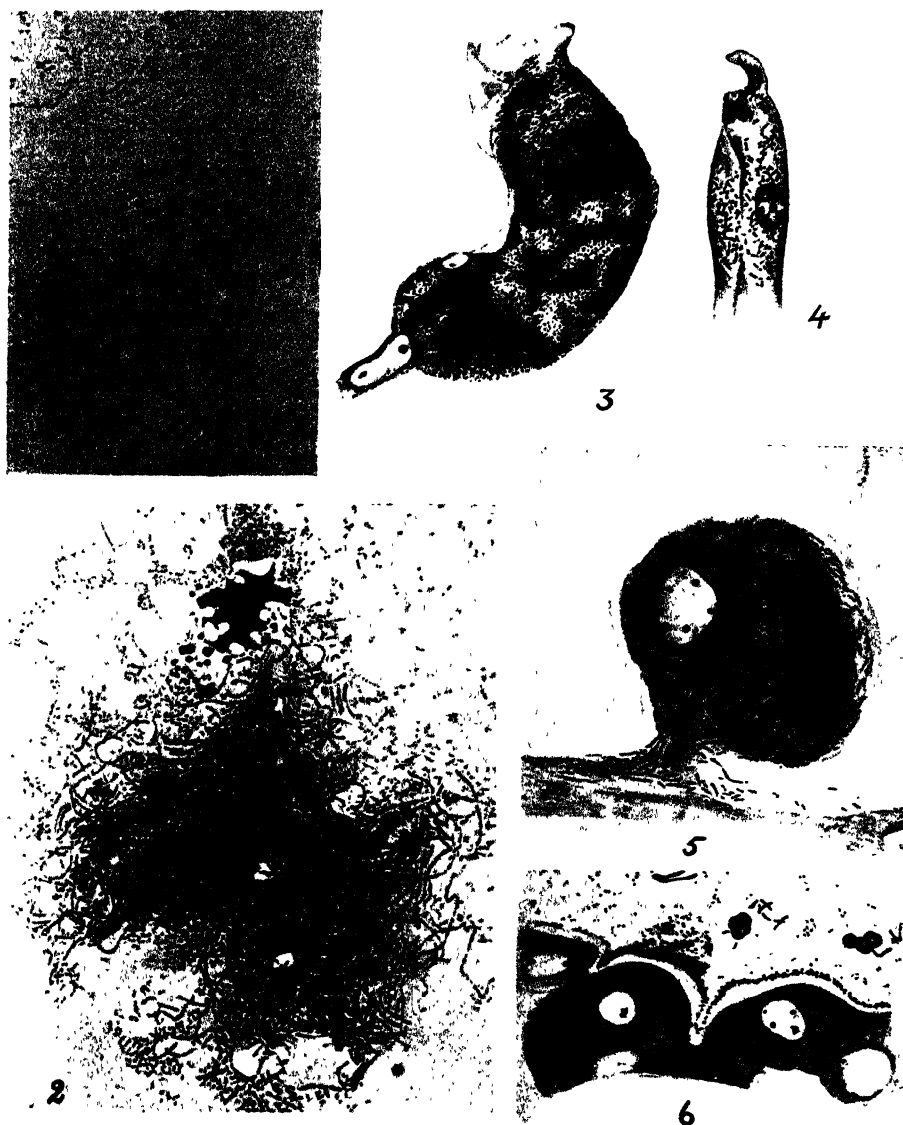
FIG. 441.—*Rickettsia* IN INTESTINE AND TISSUES OF LICE. (AFTER WOLBACH, TODD, AND PALFREY 1922.)

1. Bacillary forms of *Rickettsia prowazeki* in smear of intestinal contents ($\times$ 1,350).
2. Granular, paired, and thread-like forms of *R. prowazeki* from squashed intestinal cell ($\times$ 900).
3. Section of swollen intestinal cell containing granular mass of *R. prowazeki* at one end and chains of the same organism at the other ($\times$ 900).
4. Coccoid, diplococcoid, and rod forms of *R. prowazeki* in section of intestinal cell ($\times$ 900).
5. Section of swollen intestinal cell containing bacillary forms of *R. prowazeki* ($\times$ 1,350).
6. Section of intestinal wall showing masses of *R. pediculi* on the surface of, but not within the intestinal cells ($\times$ 900).

Chowning (1904) described as being the cause of the disease *Piroplasma hominis*, an organism which they said occurred in the blood of human cases and was similar to that causing Texas fever of cattle. Further investigations by Stiles (1905) and others showed that no such organism exists. Wolbach (1919), to whose paper reference can be made for a complete account of the disease, demonstrated changes in the endothelial cells of the vessels similar to those found in typhus fever, and the presence in these cells of bodies like *Rickettsia*. The same structures were also found in the ticks. They differ in some respects from *R. prowazeki* of typhus fever, and the name *Dermacentroxenus rickettsi* is proposed for them.

Rickettsia-like bodies have not only been discovered in non-infective lice, but have also been seen in bed bugs (*Cimex lectularius*) by Arkwright, Atkin and Bacot (1921), who named them *R. lectularia* (Fig. 442, B). Another form was seen by them in *Cimex hirundinis*, while one was met with in the sheep ked (*Melophagus ovinus*) by Nöller (1917), who gave it the name *R. melophagi* (Fig. 442, A). A supposed form in the cat flea was called *R. ctenocephali* by Sikora (1918). Hindle (1921) again saw rickettsias in lice which had fed upon healthy persons only. This form he named *R. pediculi*. It is probably identical with the *R. pediculi* of Munk and Rocha-Lima (1917), and the *R. rocha-limæ* of Weigl (1921). Another form in the melophage (*Trichodectes pilosus*) of the horse Hindle named *R. trichodectæ*, and one in the goat louse (*Linognathus stenopsis*) he called *R. linognathi*. Cowdry (1923) has described a number of forms from ticks and insects. Knowles, Napier and Smith (1924) observed Rickettsia-like bodies in the sand fly (*Phlebotomus minutus*) in India. According to Hertig and Wolbach (1924), who have reviewed all published records, over forty Rickettsia-like organisms have been reported.

These various structures which have been described are extremely minute, the rod-like forms being hardly 1 micron in length, so that accurate observations on their structure are exceedingly difficult to make. In many cases, observers have been unable to distinguish between the true rickettsias and cell granules, so that assertions as to the nature of the organisms are of little value. It is probable that some, at any rate, of the structures represent parasites, but whether these are bacteria or not cannot be definitely asserted. There is certainly no evidence in favour of their being Protozoa. Nöller (1917) claims to have cultivated the form which occurs in *Melophagus ovinus*, and Nöller and Kuchling (1923) one from the blood of sheep. Hertig and Wolbach (1924) also report the successful culture of *R. melophagi*.

Cowdry (1925), in South Africa, has shown that a disease of cattle, sheep, and goats, which is known as heartwater from the fact that the most characteristic lesion is hydropericardium, is due to the presence of a *Rickettsia* (*R. ruminantium*) in the endothelial cells of the small bloodvessels, particularly those of the renal glomeruli and cerebral cortex. Furthermore, the disease is transmitted by ticks (*Amblyomma hebræum*), and their infectivity is associated with the presence in the intestines of parasites which are morphologically identical with those in the vertebrates. Experimenting with ticks hatched from eggs, it was found that only those which had been allowed to feed on infected animals harboured the parasite and were capable of transmitting the infection.

Though many different species of *Rickettsia* have been named, authors are by no means agreed as to their nature. Woodcock (1923) believes that they represent merely granules which result from disintegration or lysis of red blood-corpuscles or other cells. It seems not impossible that in some cases granules described as *Rickettsia* may have originated in this way, and that in others actual organisms are involved. The recent work of Reichenow (1922) on intracellular symbionts in

blood-sucking mites and leeches may throw light on the whole question. Cowdry (1923) defined *Rickettsia* as including Gram-negative, intracellular, bacterium-like organisms.

A B
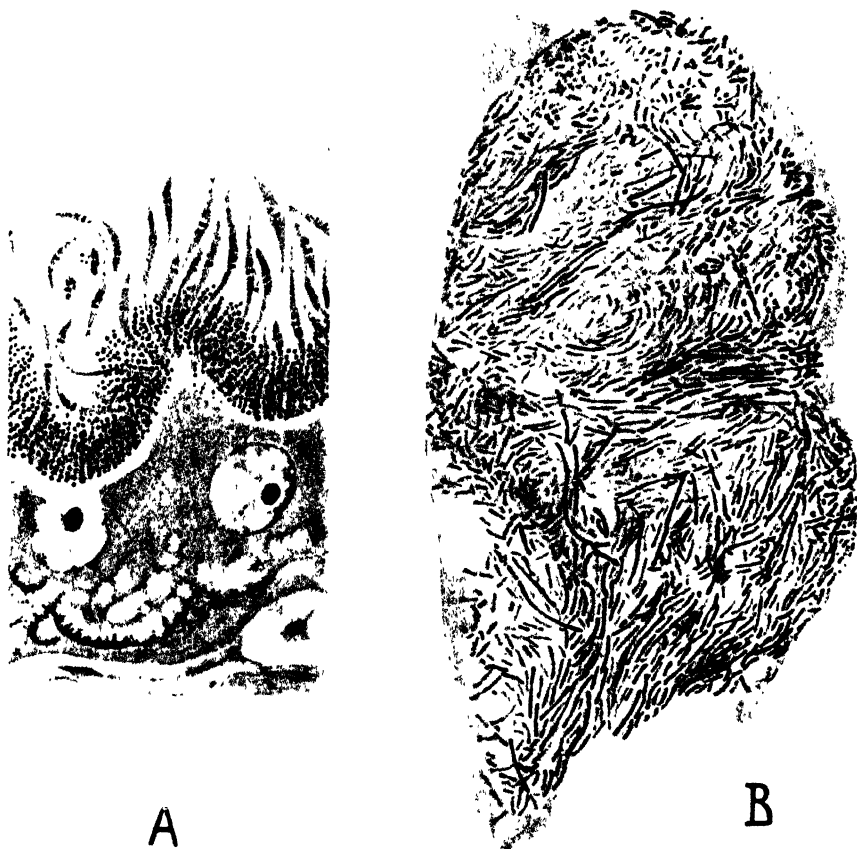
FIG. 442. —*Rickettsia* IN THE SHEEP KED AND BED BUG (× 1,500). (AFTER HERTIG AND WOLBACH, 1924.)

A. Section of intestine of sheep ked showing attached flagellates (*Trypanosoma melophagium*) and coccoid forms of *Rickettsia melophagi*.
B Cell of Malpighian tube of *Cimex lectularius* filled with filamentous, coccoid, and rod forms of *Rickettsia lectularia*.

Paraplasma SEIDELIN, 1912.

Seidelin (1912) described certain minute bodies which he had found in very scanty numbers in the red blood-corpuscles of yellow fever cases. These were supposed to consist of cytoplasm and chromatin, and to be related to the piroplasmata. He concluded that he had discovered the causative agent of yellow fever, and gave to the organism the name of *Paraplasma flavigenum*. He claimed to have transmitted it to guinea-pigs, and to have discovered naturally infected guinea-pigs in West Africa, which were supposed, therefore, to be reservoirs of the disease.

During the following years much discussion on the nature of these bodies took place, some regarding them as parasites, others as nuclear remnants or even artifacts. The writer and Low (1914) finally refuted the parasitic view, showing that exactly similar bodies occurred in normal guinea-pigs in England, and especially in young animals. It was also shown that they occurred in human beings in anæmic conditions. It seems that some of the bodies described as *P. flavigenum* represent structural irregularities in the red cells dependent on the presence of nuclei at the early stages of their development, while others are undoubtedly artifacts.

Microsoma LEBEDEFF AND TSCHARNOTZKY, 1911.

Certain structures in the red blood-corpuscles which were devoid of pigment were described by Lebedeff and Tscharnotzky (1911) from the Russian polecat, *Putorius putorius*, under the name of *Microsoma mustelæ*. Amœboid forms were seen, as also large globular parasites which nearly or completely filled the red blood-corpuscles. The parasites were peculiar in having very small nuclei. No details of the development were elucidated, and there is very little evidence that the bodies were organisms.

Cingula AWERINZEW, 1914.

Under the name of *Cingula boodontis* Awerinzew (1914) described a minute organism, if indeed it is a parasite at all, from the red cells of the snake, *Boodon lineatus*. It occurred as a small granule surrounded by a clear area. In later stages a vacuole appeared in it and converted it into a ring, on one side of which a nucleus could be seen. Division into two is then said to take place. What may possibly be the same structures were described by Johnston (1917) from the blood of two snakes (*Echis carinatus* and *Causus rhombeatus*) of West Africa. He described two types of body, one which was homogeneous and took a blue colour after Giemsa or Leishman stain, while the other was granular and stained red. The writer has seen similar structures in the blood of snakes, but can produce no evidence as to their parasitic nature.

Immanoplasma NEUMANN, 1909.

Neumann (1909) described a parasite of the red cells of the dog-fish, *Scyllium canicula*. It was devoid of pigment, and occurred as masses of cytoplasm varying in size up to 30 by 20 microns. Two types were recognized: male forms with faintly staining cytoplasm and a large nucleus, and female forms with deeply staining cytoplasm and small nucleus. In life the organism is feebly amœboid. The red cell is considerably distorted by the parasite. Nothing is known of its life-history.

Globidiellum BRUMPT, 1913.

Neumann (1909) intended to found a new genus, *Globidium*, for a parasite which he discovered in the mononuclear leucocytes or endothelial cells of the haddock (*Gadus æglefinus*). As this name had been given to a totally distinct parasite (see p. 769), Brumpt (1913c) proposed the name *Globidiellum* (Fig. 443, 7-10). The parasite will be considered below (p. 1107) as possibly representing a stage in the life-cycle of a hæmogregarine.

Hæmatractidium HENRY, 1910.

Henry (1910) described a parasite, which did not produce pigment, in the red cells of the common mackerel (*Scomber scomber*). A detailed description of the organism was given in 1913 (Fig. 443, 1-6). The youngest stage figured is a minute

cytoplasmic body containing two chromatin granules. As increase in size takes place the parasite elongates, and finally occupies the cell on one side of the nucleus. The largest forms are slightly longer than the nuclei of the host cell, and have one end rounded and the other tapering. The nucleus of the parasite always remains small. The organism is supposed to have a remarkable destructive action on the red cell, which quickly degenerates and eventually liberates the parasites. The figures illustrating the breaking-down of the cell rather suggest a mechanical damage in film-making than a process brought about by the parasite.
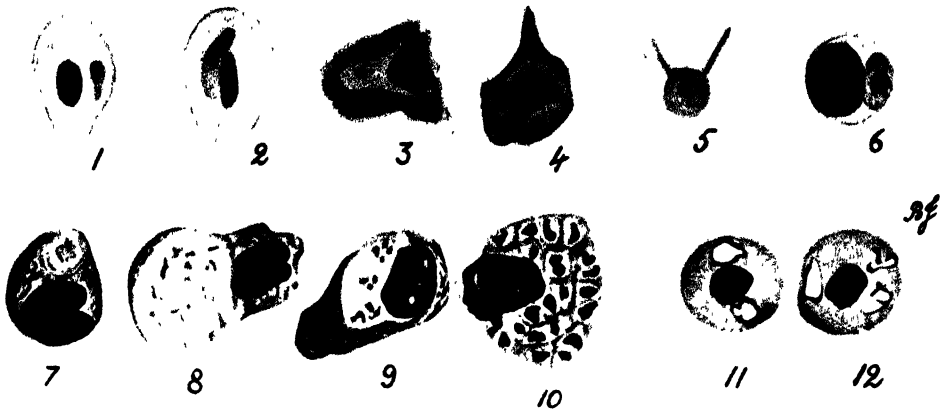
FIG. 443.—QUESTIONABLE STRUCTURES SEEN IN BLOOD-CORPUSCLES OF MARINE FISH (× ca. 1,500). (AFTER HENRY, 1913.)

1-6. *Hæmatractidium scombri* from the common mackerel (*Scomber scomber*).
7-10. *Globidiellum multifidum* of Neumann, thought by Henry to represent the schizogony of *Hæmogregarina æglefini* of the haddock (*Gadus æglefinus*).
11-12. Structures in the blood-corpuscles of the sole (*Solea vulgaris*), which are supposed to be derived from granules extruded by hæmogregarines. They were previously seen by Henry in other fish and named *Hæmohormidium cotti*.

Hæmohormidium HENRY, 1910.

This parasite was described by Henry (1910 and 1913b) as occurring in the red cells of two marine fish, *Cottus bubalis* and *Cottus scorpius*. It is an irregularly round or oval body, and varies in length from 2 to 4·5 microns (Fig. 443, 11-12). The chromatin is arranged along the margin, about two-thirds of which it occupies. As many as three of the organisms were found in a single cell. They bear some resemblance to ring forms of malarial parasites. Henry came to the remarkable conclusion that they represented the intracorpuscular stages of " infective granules " extruded by hæmogregarines.

Possible Pigment-Free Parasites of the Red Blood-Corpuscles of Birds.

In connection with the spirochætal disease of fowls in various parts of the world peculiar bodies are sometimes seen in large numbers within the red blood-corpuscles. It has been suggested that they represent an intracellular granular phase of the spirochæte. On the other hand, it may be that a distinct organism is represented, though the possible origin of the bodies from extruded portions of the cell nuclei has not been excluded. They are referred to more fully below (p. 1262).

B. *Order:* ADELEIDA.

1. *Sub-Order:* Adeleidea LÉGER, 1911.

This sub-order, which has been defined above (p. 794), can be sub-divided into a number of families and sub-families as follows:

1. *Family:* DOBELLIIDÆ.—The oöcyst contains a number of sporozoites without sporocysts. There are two lines of schizogony, one terminating in the production of microgametocytes and the other macrogametocytes. Association of these occurs as in other members of the sub-order, but the microgametocyte produces a large number of microgametes instead of the usual small number. The zygote becomes encysted in the oöcyst, and sporozoites are produced. This form holds an intermediate position between the sub-orders Eimeriidea and Adeleidea in that it conforms with the former in the production of numerous microgametes and with the latter in the close association of the two gametocytes prior to fertilization.

2. *Family:* LEGERELLIDÆ Léger, 1911.—The oöcyst contains a number of sporozoites, but no sporocysts. The schizogony cycle is described as running on two lines, one producing a type of merozoite which will ultimately become microgametes, and the other a second type of merozoite leading to formation of macrogametes.

3. *Family:* ADELEIDÆ Mesnil, 1903.—The oöcyst contains sporocysts, which include sporozoites. There are three sub-families.

(1) *Sub-Family:* ADELEINÆ.—The oöcyst is spherical and contains a variable number of sporocysts, each containing two sporozoites. The sporocysts are disc-like, and resemble two apposed watch-glasses.

(2) *Sub-Family:* KLOSSIINÆ.—The spherical oöcyst contains many spherical sporocysts, each of which includes four sporozoites.

3. *Sub-Family:* CHAGASELLINÆ.—The oöcyst contains only three sporocysts, which contain four, six, or more sporozoites.

4. *Family:* KLOSSIELLIDÆ.—The spherical oöcyst produces a number of spherical sporocysts, each of which contains numerous sporozoites. Schizogony takes place in the usual manner with production of merozoites. Finally, certain merozoites take up another situation in the host, and undergo, after growth, another type of schizogony, producing merozoites which grow into gametocytes. The male gametocyte produces only two microgametes.

These families may now be considered in greater detail.

SYSTEMATIC DESCRIPTION OF THE FAMILIES OF THE SUB-ORDER ADELEIDEA.

1. *Family :* DOBELLIIDÆ.

This family includes the single genus *Dobellia* founded by Ikeda (1914) for the only known species, *Dobellia binucleata*, a gut parasite of the sipunculoid worm, *Petalostoma minutum.* Many of the stages are described as containing two nuclei. In the behaviour of its gametocytes it differs from other members of the sub-order Adeleidea in that the microgametocyte, though it enters into syzygy with the macrogametocyte, produces a large number of microgametes. In this respect it may be regarded as a connecting link between the two sub-orders of the Coccidiomorpha. The infection commences by sporozoites liberated in the gut from the oöcyst. Some of the sporozoites enter the cells and penetrate their nuclei, where they commence growing. When partially grown, they leave the nuclei and continue their development in the cytoplasm. When fully grown, they break up into a number of large merozoites, which enter other cells and repeat the process of schizogony. Other sporozoites do not enter the cells, but attach themselves to their surfaces and develop into smaller schizonts, which produce small merozoites. Eventually, large merozoites within the cytoplasm develop into macrogametocytes. Meanwhile, some of the small merozoites produced on the surface of the cells enter the cytoplasm and develop into microgametocytes in close association with the macrogametocytes. Each microgametocyte produces a large number of microgametes, one of which fertilizes a macrogamete. An oöcyst is formed measuring from 20 to 25 microns in diameter, and within it are produced, without sporocysts, about 100 sporozoites. The sporozoites resemble those found in the cytoplasm of the gut cells, and presumably develop into the large schizonts. Ikeda has noted that another type of oöcyst may be present, and that it differs from the others in being only half the size and in producing a smaller number of sporozoites. It is suggested that the sporozoites from the smaller oöcysts become the small schizonts which develop on the surface of the gut epithelium. If this be so, then sexual dimorphism not only differentiates the schizogony cycle, but extends to the oöcysts and sporozoites. It is difficult to accept these claims till further confirmation has been obtained. The name *D. binucleata* was given to the parasite on account of a second body, considered to be of nuclear nature, which was associated with the nucleus in many stages of the development. Dobell (1925), in whose laboratory Ikeda made his observations, admits that Ikeda may have misinterpreted certain stages, but is convinced that the parasite is binucleate, one of the nuclei being somatic and the other germinal.

2. *Family:* LEGERELLIDÆ Léger, 1911.

This family includes the single genus *Legerella*, which was founded by Mesnil (1910), for a parasite which Aimé Schneider (1881) described as *Eimeria nova* from the Malpighian tubes of the myriapod *Glomeris* sp. This form was studied by Léger, L. (1900*b*), while Cuénot (1902) described
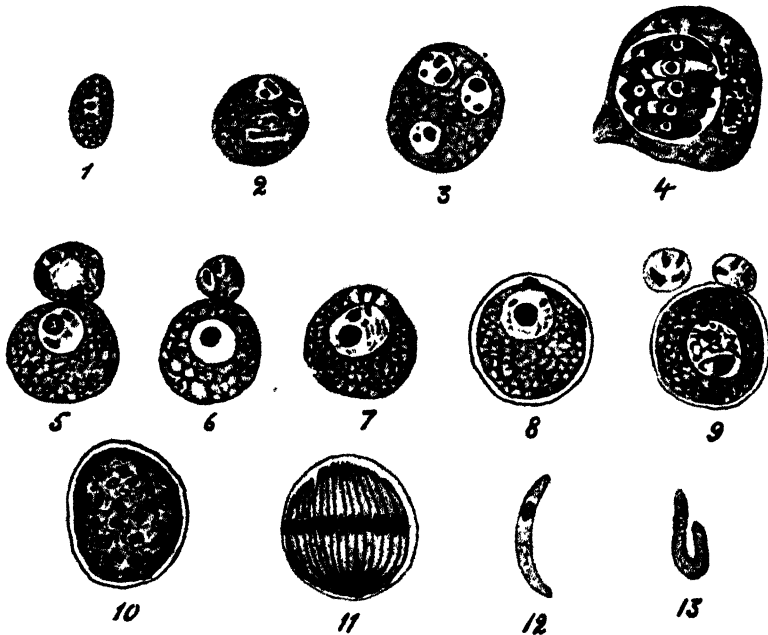
FIG. 444.—*Legerella parva* FROM THE MALPIGHIAN TUBES OF THE DOG FLEA (× 1,030).
(AFTER NÖLLER, 1914.)

1-4. Schizogony cycle.
 5. Associated macrogametocyte and microgametocyte which is producing four microgametes.
6-7. Fertilization.   8. Oöcyst
 9. Oöcyst with two attached microgametocytes, one with four and the other with three microgametes. One microgamete has presumably fertilized the macrogamete
10. Oöcyst with multinucleated sporont.
11. Mature oöcyst with sporozoites and residual body.   12-13. Sporozoites.

as *L. testicula* another which occurred in the testes of *Glomeris*. Nöller (1913) gave the name *L. parva* to a parasite of the Malpighian tubes of chicken and pigeon fleas (*Ceratophyllus gallinæ* and *C. columbæ*), 25 to 40 per cent. of which were infected. A more detailed account was published by him later (1914). The development of the parasite, which occurs only in the adult fleas, takes place in the cells of the Malpighian tubes (Fig. 444). The merozoites measure from 6 to 8 microns in length

by 2 to 4 microns in breadth, and these increase in size till they have a diameter of 10 to 15 microns. Nuclear multiplication takes place by repeated divisions till eight to twenty are present. The schizont then breaks up into a corresponding number of merozoites. Eventually certain merozoites become gametocytes. The microgametocyte is not much larger than the merozoite, but it is closely applied to the large macrogametocyte, which has a diameter of 16 to 20 microns. The microgametocyte produces four microgametes, one of which fertilizes the macrogamete. An oöcyst is formed, and within it the zygote breaks up into sporozoites, of which as many as sixty may be present. The sporozoite is a long, narrow structure measuring 20 by 2 to 3 microns. It is probable that the oöcysts escape into the gut after passing down the Malpighian tubes, and are voided in the fæces of the flea. They are then probably taken up by flea larvæ, the infection passing through the pupal stage to the adult flea. Though Nöller could not find an infection in the larva, fleas taken from the pupal cases were found infected, so that the infection must have been present in the larval stage.

Splendore (1920) recorded another species (*L. grassii*) from the Malpighian tubes of *Ceratophyllus fasciatus*.

3. *Family:* ADELEIDÆ Mesnil, 1903.

This family comprises the three sub-families *Adeleinæ*, *Klossiinæ*, and *Chagasellinæ*, which differ from one another in the number of sporozoites within the sporocyst.

(1) *Sub-Family:* ADELEINÆ.

This sub-family includes the two genera *Adelea* and *Adelina*, which are characterized by the large oöcysts containing a variable number of sporocysts, each with two sporozoites.

Genus: **Adelea** Aimé Schneider, 1875.

This genus was founded by Schneider (1875) for the coccidium *Adelea ovata*, which has been described above as a type of the sub-order *Adeleidea* (Fig. 338). Another form which closely resembles it is *A. mesnili* Perez, 1903, which is parasitic in the cœlom of the lepidopteran *Tineola biseliella*. The parasite described as *A. hartmanni* by Chagas (1910) was placed by Machado (1911a) in the genus *Chagasella*, considered below. The coccidium named *A. pachylabræ* by de Mello (1921), which occurs in the Indian mollusc, *Pachylabra mœsta*, has an oöcyst with two sporocysts, each with two sporozoites. It is doubtful if it belongs to this genus.

Genus : Adelina Hesse, 1911.

This genus was founded by Hesse (1911) for a coccidium of the oligochæte worm, *Slavinia appendiculata.* It was named *A. octospora* and placed in a new genus because the sporocysts differed from those of members of the genus *Adelea* in that they were present in the oöcyst in small numbers, and were spherical instead of discoidal. He placed in this new genus several species which had hitherto been considered as belonging to the genus *Adelea* (*A. zonula, A. transita, A. akidum,* and *A. dimidiata*). Another form which must be included is the one described by Léger and Duboscq (1902) as *A. dimidiata* var. *coccidioides.*

Adelina dimidiata (Aimé Schneider, 1885).—This coccidium was first seen by Aimé Schneider (1885) in *Scolopendra morsitans,* and named by him *Klossia dimidiata.* It was rediscovered by Balbiani (1889), and studied by Léger (1897, 1898), who found it in *Scolopendra cingulata* and *S. subspinipes.* Léger and Duboscq (1902) found a closely allied form (*A. dimidiata coccidioides*) in *S. oraniensis lusitanica.* Finally, Schellack (1913) gave a detailed account of the development of *A. dimidiata* in *S. cingulata.* Its life-history resembleᴗ that of *A. ovata.* The merozoites vary in length from 3 to 21 microns and in breadth from 1·8 to 2·5 microns, while the number produced at any schizogony varies from four to thirty (Fig. 445, A-C). Léger and Duboscq (1902) stated that there were two distinct types of schizogony, the merozoites from one of which became either schizonts again or microgametocytes, while those from the other became macrogametocytes. No such dimorphism could be detected by Schellack. The macrogametocyte can first be recognized by its size and the lack of nuclear multiplication characteristic of the schizont. As it increases in size, one end of the elongated macrogametocyte becomes differentiated as a finger-like process, which becomes longer at later stages of development (Fig. 445, D). The nucleus retains its karyosome throughout. The young microgametocyte cannot be distinguished from a young schizont except for the fact that it very soon becomes associated with a growing macrogametocyte, as in *A. ovata.* It also becomes drawn out into a finger-like process. The presence of these finger-like processes on the gametocytes is one of the characteristic features of the parasite. Eventually they are withdrawn, and a fine membrane is formed around the two gametocytes (Fig. 445, E). At this stage the paired gametocytes within the enclosing membrane fall out of the epithelial cell into the lumen of the intestine. By repeated division of the nucleus of the microgametocyte four nuclei are formed. These come to the surface and give rise to four microgametes. The microgametes then become free within the enclosing common membrane, and pass to the opposite pole

of the macrogamete, where its nucleus has moved.   Penetration is effected
by one of the microgametes, the nucleus of which fuses with that of the
macrogamete.   Nothing which could be interpreted as a process of matura-
tion of the macrogametocyte nucleus which retains the karyosome could
be detected.   After fertilization, an oöcyst 0·5 to 1 micron in thickness
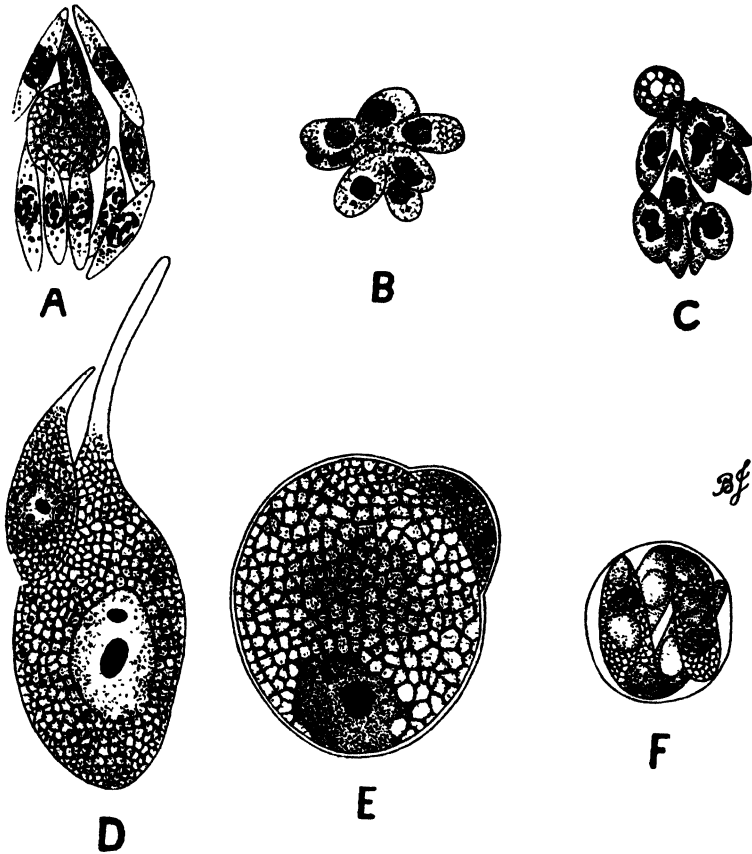
FIG. 445.   *Adelina dimidiata* (× 1,700).   (AFTER SCHELLACK, 1913.)

A-C Three schizonts showing variations in the size and the number of merozoites.
   D. Associated macro- and micro-gametocytes with digital processes.
   E. The digital processes have been withdrawn.   The microgametocyte has four micro-
       gamete nuclei, while the nucleus of the macrogametocyte has occupied a terminal
       position in preparation for fertilization.
   F. Sporocyst with the two sporozoites.

is secreted round the zygote, and it appears that within it are included
the remains of the microgametocyte and the three unused microgametes.
At the pole opposite to that at which the remains of the microgametocyte
lie can often be detected a small circular suture, which is probably a
micropyle.   Within the oöcyst there are formed a number of sporoblasts

by a budding process which leaves a large residual body. The sporoblasts secrete spherical sporocysts, within each of which are developed two sporozoites without formation of a residual body. Within the sporocyst the two sporozoites are twisted round one another in a characteristic manner (Fig. 445, F). By the time the sporocysts have developed the residual body within the oöcyst has disintegrated. The size of the oöcysts depends on the number of sporocysts, which varies from three to seventeen. The sporocysts have a diameter of 15 microns. The period required for complete development of the oöcyst is about seven days.
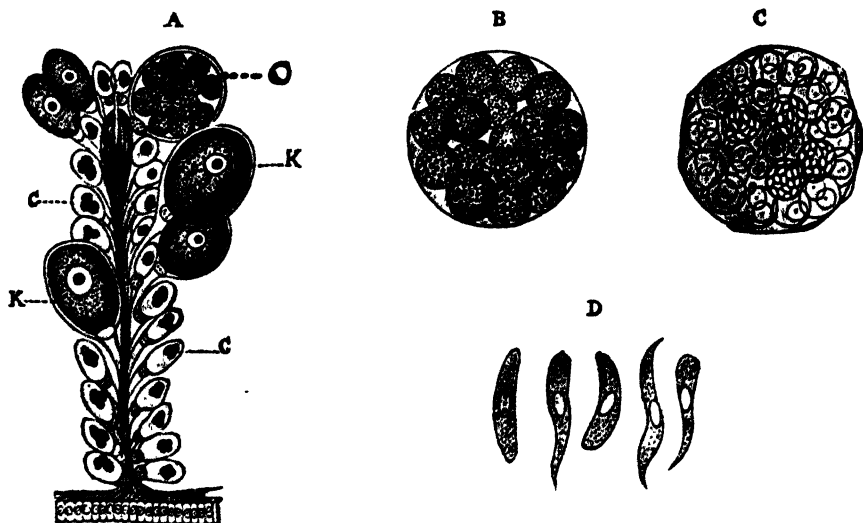
FIG. 446.—*Klossia helicina* FROM THE KIDNEY OF THE SNAIL, *Helix hortensis*.
(AFTER BALBIANI, 1884.)

A. Section of the kidney showing C, healthy epithelium with included concretions; K. oöcysts with contained zygotes in hypertrophied cells; and O, oöcyst with numerous immature sporocysts ($\times$ *ca.* 120).
B. Oöcyst with immature sporocysts more highly magnified ($\times$ *ca.* 240).
C. Oöcyst with mature sporocysts, each with four sporozoites and a residual body ($\times$ *ca.* 240).
D. Free sporozoites.

Schellack does not believe that the form *A. dimidiata coccidioides* described by Léger and Duboscq (1902) can be regarded as distinct from *A. dimidiata*. Other species described are *A. akidum* Léger, 1900, from *Olocrates abbreviatus*; *A. transita* Léger, 1904, from *Embia soleri*; *A. zonula* Moroff, 1906, from *Blaps mortisaga*; and *A.* sp. Chatton, 1912, from *Scincus officinalis*.

(2) *Sub-Family:* KLOSSIINÆ.

This sub-family includes forms which have oöcysts containing a large number of sporocysts, each of which has four sporozoites. There are two

genera, *Klossia* and *Orcheobius*. The genus *Klossia* was founded by Aimé Schneider (1875a) for a parasite which Kloss (1855) had discovered in the kidney of the land snail. The genus *Orcheobius*, which differs from *Klossia* in that the gametocytes are elongate instead of being spherical, was established by Schuberg and Kunze (1906) for a parasite of the testis of the leech, *Herpobdella atomaria*.
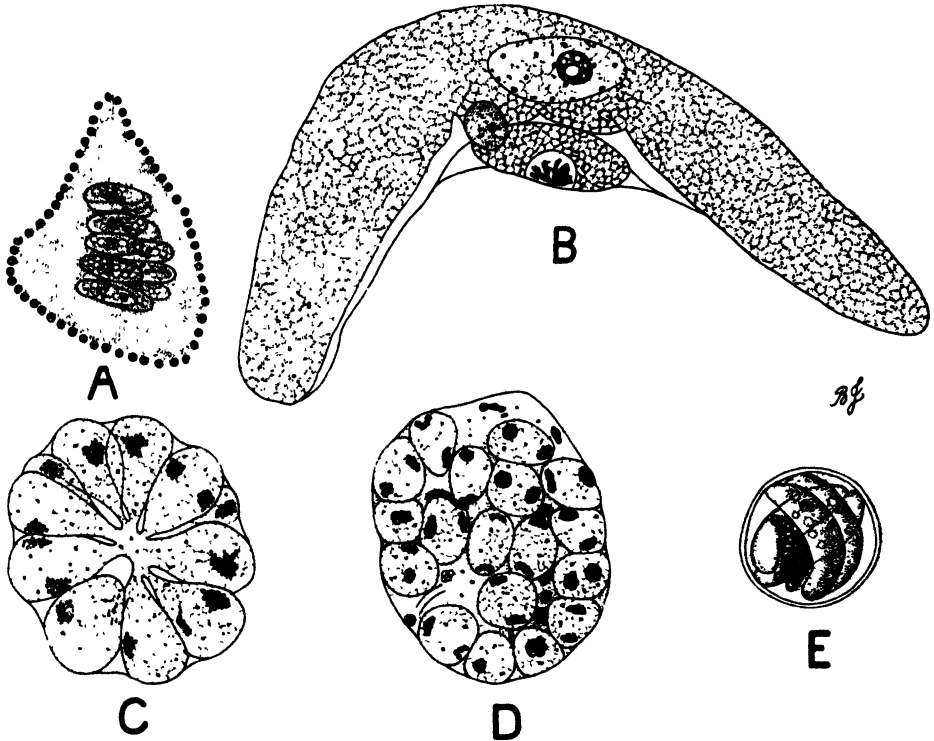
FIG. 447.—*Orcheobius herpobdellæ* PARASITIC IN THE TESTIS OF THE LEECH, *Herpobdella atomaria*. (AFTER KUNZE, 1907.)

A. Schizogony within a cytophore of the testis (× 800).
B. Associated macro- and micro-gametocyte (× 400)
C. Formation of sporoblasts within the oöcyst (× 800).
D. Oöcyst containing developing sporocysts (× 400).
E. Fully-developed sporocyst with four sporozoites and residual body (× 800).

Klossia helicina Ai. Schneider, 1875.—This coccidium, which is a common parasite of various land snails, was first seen by Kloss (1855), who gave an excellent description of the parasite. It was the first occasion on which anything like a complete account of the life-history of a coccidium had been published. It was studied by Schneider (1875a) and Balbiani (1884) in *Helix hortensis*, and by L. Pfeiffer (1890) in this snail, as well as *Succinea pfeifferi*, *H. nemoralis*, and *H. arbustorum*. Laveran (1898) found it

in *H. hortensis*, and gave an account of the main features of its life-history, but it was Debaisieux (1911) who first pointed out that there occurred an association of the micro- and macro-gametocytes, and that the former gave rise to four microgametes, as in *Adelea ovata*. Moroff (1911) gave a similar account for a parasite of *Vitria elliptica*. He named it *Klossia vitrina*, but it is not clear that it is distinct from *K. helicina*, which has also been recorded from *Helix hispida, H. practicum, H. umbrosa*, and *Succinea gigantea*.

The life-cycle of *K. helicina* is very similar to that of *A. ovata*. It develops in the epithelial cells of the kidney (Fig. 446). The oöcysts, which are usually spherical, vary in diameter from 40 to 180 microns. The number of sporocysts, which are spherical, vary in number according to the size of the oöcyst. In the largest cysts there may be as many as 160. Each sporocyst contains four sporozoites and a residual body. Exceptionally there may be a larger number of sporozoites present.

Orcheobius herpobdellæ Schuberg and Kunze, 1906.—This form, which is the only representative of the genus, was discovered by Schuberg and Kunze (1906) in the testis of the leech, *Herpobdella atomaria* (*Nephelis vulgaris*). Its most characteristic feature is the peculiar shape of the associated micro- and macro-gametocytes, which are much elongated (Fig. 447). The former measure about 50 by 12 microns, and the latter 180 by 30 microns. The microgametocyte gives rise to four microgametes. The spherical oöcyst contains twenty-five to thirty spherical sporocysts, each of which has typically four sporozoites. During schizogony from twelve to twenty merozoites are produced.

(3) *Sub-Family :* CHAGASELLINÆ.

This sub-family contains the single genus *Chagasella* Machado, 1911. It was established for a parasite which Chagas (1910) had described under the name *Adelea hartmanni* from the intestine of the Brazilian bug, *Dysdercus ruficollis*. Machado (1911a) placed it in a new genus, *Chagasella*, as the name *Chagasia* proposed by Léger (1911) was preoccupied. Machado (1913) described another species, *Chagasella alydi*, from another bug, *Alydus* sp.

Chagasella hartmanni (Chagas, 1910).—This is an intestinal parasite of the bug, *Dysdercus ruficollis*. It develops in the epithelial cells, and, according to Chagas, reproduces there by two types of schizogony, one of which gives rise to about ten to twenty large merozoites (macroschizogony), and the other to about the same number of small merozoites (microschizogony) (Fig. 448, A and B). The male and female gametocytes develop in association with one another, and, as sometimes occurs in the case of *Adelea ovata*, several microgametocytes may be associated with one

macrogametocyte. Each microgametocyte gives rise to four micro-gametes (Fig. 448, C). The oöcyst gives rise to only three sporoblasts, which become three sporocysts, in each of which four sporozoites are developed (Fig. 448, D and E). The second species, *Chagasella alydi* Machado, 1913, is parasitic not only in the intestine, but also in the generative organs of its host. It resembles *C. hartmanni* in the main
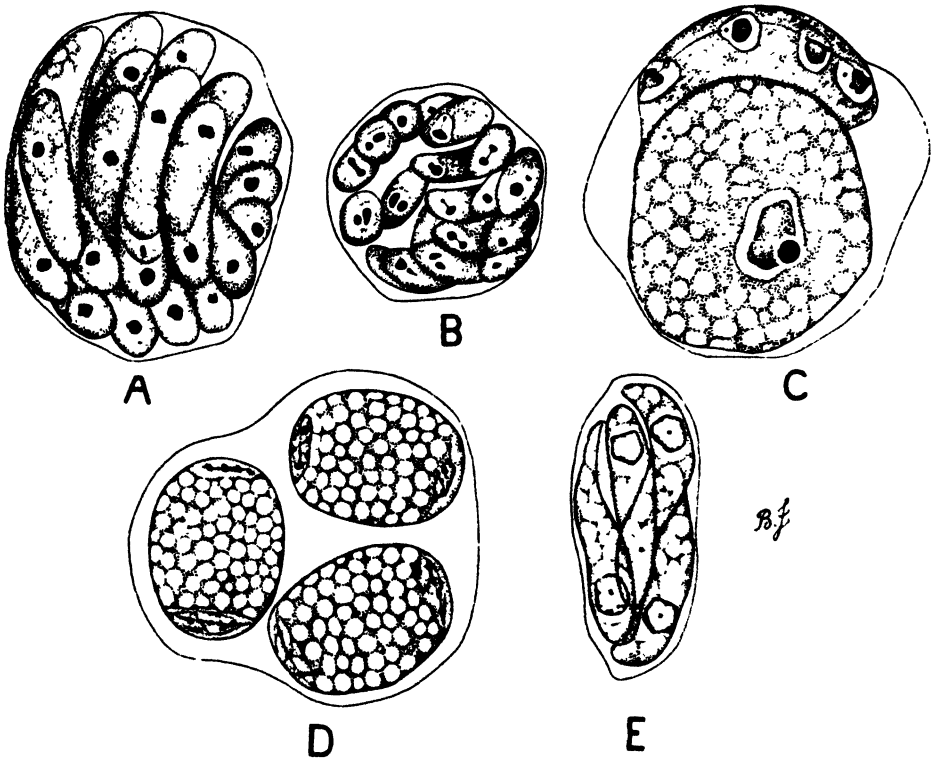
FIG. 448. *Chagasella hartmanni* PARASITIC IN THE INTESTINE OF THE BUG, *Dysdercus ruficollis* (× *ca*. 1,000). (AFTER CHAGAS, 1910.)

A-B. Merozoites resulting from schizogony, the so-called macro- and micro-schizogony.
C. Macrogametocyte with associated microgametocyte with four microgamete nuclei.
D. Oöcyst with three sporoblasts. E. Sporocyst with four sporozoites.

features of its life-history, but differs in that six and sometimes more sporozoites are produced within the oöcyst.

It seems very doubtful if the two types of schizogony are as sharply marked off from one another as Chagas and Machado maintain, for in forms like *Adelea ovata* and *Adelina dimidiata*, described above, the number and size of the merozoites is subject to considerable variations. It seems also possible that the two species described are really a single species, with sporocysts containing a variable number of sporozoites.

4. *Family :* KLOSSIELLIDÆ.

This family includes the single genus *Klossiella*, which was established by Smith and Johnson (1902) for a coccidium which they named *K. muris*, and which is a common parasite of the kidney of mice. It had previously been seen by Smith (1889), while Pfeiffer, L. (1890), gave a short description and figure of the parasite. It was studied by Woodcock (1904a), Brugnatelli (1908), Sangiorgi (1911), and Stevenson (1915). Pianese (1901) discovered a similar form in the guinea-pig. It was rediscovered by Seidelin (1914), who named it *K. cobayæ*. Pearce (1916) gave a description of this parasite, while Sangiorgi (1916), who also studied it, proposed to name it *Klossia caviæ*. It evidently does not belong to the genus *Klossia*.

There has been considerable confusion over the various forms met with, but A. C. Stevenson has studied both *K. muris* and *K. cobayæ*, and the following description of the latter is based on still unpublished notes which he has kindly permitted the writer to use.

Klossiella cobayæ Seidelin, 1914.—This is a parasite of the kidney tubules and endothelial cells of the blood-vessels of the guinea-pig (Fig. 449). Most of its developmental stages can be seen in sections of the kidney, but schizogony forms, which occur in the endothelial cells of the capillaries of the glomeruli, can also be observed in similar situations in other organs. The sporozoites escaping into the lumen of the gut from the ingested sporocysts may be presumed to make their way to the vascular endothelium. Within the cytoplasm of the cell a small rounded body is produced, and this develops into a schizont having a diameter of about 2·5 microns. The single nucleus multiplies by repeated divisions till eight are present, after which eight merozoites are produced. They lie in a vacuole in the now enlarged endothelial cell, which bulges into the lumen of the vessel. By its rupture the merozoites, which are minute bodies measuring 2 by 1 microns, escape into the blood and invade other endothelial cells (Fig. 449, 1). Eventually, some of the merozoites enter the lumen of the tubules of the kidney and make their way to the convoluted portion, the cells of which they penetrate. Here they again grow into

1. Schizogony cycle in endothelial cells of blood-vessels of kidneys and other organs.
2. Passage of merozoite to the cell of the convoluted tubules of kidney.
3-5. Schizogony in convoluted tubules giving rise to a large number of young gametocytes, which pass down the tubules to the straight tubules or loops of Henle, where they enter the cells and associate in pairs.
6-7. Growth of female gametocyte and formation of two microgametes by smaller male gametocyte.
8. Zygote with remains of microgametocyte attached and one residual microgamete.
9. Growth of zygote (sporont) during nuclear multiplication.
10. Formation of sporoblasts by budding process.
11. Numerous sporocysts with sporozoites. The sporocysts escape in the urine and are presumably ingested by guinea-pigs.

FIG. 449.—DEVELOPMENT OF *Klossiella cobayæ* IN THE KIDNEY OF THE GUINEA PIG (× 2,000). (ORIGINAL FROM UNPUBLISHED DRAWINGS BY A. C. STEVENSON.)

[For description see opposite page.

schizonts and produce about one hundred daughter individuals, which are the young gametocytes (Fig. 449, 2-5). The infected cell is much enlarged, and almost fills the lumen of the tubule. The young gameto-cytes, after rupture of the host cell, travel down the duct till they reach the straight tubules or loops of Henle, the cells of which they infect. Here they are found typically in pairs, and as growth proceeds it is noted that one individual of each pair grows more than the other (Fig. 449, 6). The smaller one is the microgametocyte, and it becomes associated in the same vacuole with the larger macrogametocyte. The single nucleus of the microgametocyte divides once, and the two nuclei thus formed become the nuclei of the two microgametes, which are elongate structures with a central nucleus (Fig. 449, 7). Fertilization of the macrogamete takes place, and ˙a typical fertilization spindle is formed (Fig. 449, 8). It occasionally happens that two or three microgametocytes become associated with the macrogamete, but only one of the microgametes produced is employed in fertilization. The zygote increases in size, causing its host cell to swell considerably till the lumen of the tubule is completely filled (Fig. 449, 9). The growth takes place in the vacuole, and it is doubtful if a true oöcyst is formed. As growth proceeds, the nucleus multiplies till thirty or more are present. The sporont then has a diameter of about 30 to 40 microns. By a process of budding from the surface there are produced a number of sporoblasts, each of which becomes slightly elongated and encloses itself in a sporocyst. Within the sporocyst the sporoblast produces about thirty sporozoites (Fig. 449, 10-11). When fully formed, the sporocysts pass down the tubules and escape in the urine, and are ready to produce infection in another host.

Klossiella muris Smith and Johnson, 1902.—This is a common parasite of white mice, and is not infrequently encountered in sections of the kidney, where it has a cycle similar to that of *K. cobayæ*. The whole of the schizogony cycle, including the final one into young gametocytes, takes place in the endothelial cells of the capillaries of the glomeruli. The young gametocytes then enter the tubules, and their subsequent develop-ment takes place in the cells of the convoluted tubules, instead of lower down, as in *K. cobayæ*.

Hartmann and Schilling (1917) described certain stages of development of *Hepatozoon balfouri*, the hæmogregarine of the red blood-corpuscles of the jerboa, as taking place in the kidney. Reichenow (1921a) states that Nöller informed him that these were stages of development of a species of *Klossiella*. This parasite of the jerboa was seen by the writer in the Sudan in 1906, and he agrees with Nöller that the parasite is not *H. balfouri*, but a *Klossiella* of the jerboa, which may or may not be

a species distinct from that of the mouse. The only forms seen appear to be the schizogony stages in the tubules.

Pneumocystis carinii Delanoë, 1912. This organism, which was named by Delanoë (1912), possibly represents the schizogony stage of species of *Klossiella*, which, as pointed out above, occurs not only in the endothelial cells of the blood-vessels of the glomeruli of the kidney, but also in those of the vessels of other organs, such as the lung (Fig. 450). It is found, however, in animals, such as the dog, which are not known to harbour *Klossiella*.

In smears of the lung of guinea-pigs and other animals it is seen as small round cysts containing eight uninucleate bodies. They were first described by Chagas (1909), who found them in smears of the lungs of guinea-pigs infected with *Trypanosoma cruzi*. Carini (1910*a*) found identi-
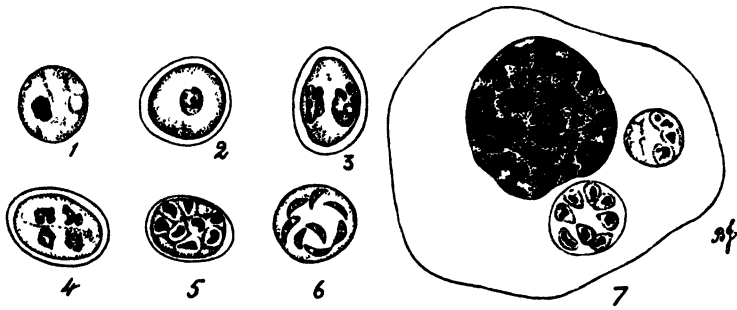
FIG. 450.—*Pneumocystis carinii* FROM THE LUNG SMEARS OF A DOG (× 2,000). (AFTER CARINI AND MACIEL, 1914.)

1-6. Various stages in development.
7. Large mononuclear cell with two included schizonts.

cal cysts in the lungs of rats infected with *T. lewisi*, and Vianna (1911*a, b*) in guinea-pigs infected with various pathogenic trypanosomes. All these observers considered the cysts to represent a reproductive phase of the trypanosomes. Delanoë (1912) studied these bodies in Paris rats, and found they had no connection with the trypanosomes, and named them *Pneumocystis carinii*. Later (1914) he found them in guinea-pigs, but failed to discover them in rabbits. They undoubtedly represent the schizogony stage of some sporozoon, and Stevenson suggests they may be schizonts of *Klossiella*, which, as shown above, develop in the endothelial cells of the vessels of other organs than the kidney. It is possible that the sporozoites, after escape from the sporocysts which have been eaten by the mouse or guinea-pig, pass through the wall of the gut and enter the first endothelial cells they reach, and that it is only after repeated schizogony that the merozoites finally spread as far as the kidney, where

it is necessary for them to arrive in order that sporogony shall occur. As a result of Delanoë's work, Chagas (1913) and Carini and Maciel (1914) admit that *P. carinii* has no connection with the trypanosome cycle. The last-named observers found a dog heavily infected. The organism was seen also by Aragão (1913), Coles (1914), and Porter (1916). Aragão found them in the rabbit, guinea-pig, and rat. He states that they were also seen by Machado in the wild guinea-pig, goat, and sheep. Porter discovered them in mice and Coles in rats in England. Aragão pointed out the resemblance of these cysts to certain stages of *Cryptosporidium muris*, the coccidium of the glands of the stomach of mice (Fig. 340).

2. *Sub-Order:* Hæmogregarinidea.

The researches of Reichenow and others during the past ten years have demonstrated the coccidial nature of a group of parasites which were recognized as occurring in red blood-corpuscles or leucocytes of vertebrate animals. These have long been known under the name of hæmogregarines, but they are now known to be in reality coccidia, which have certain stages adapted to life within the circulating cells of the blood. In certain cases (Lankesterellidæ) the life-cycle is typical of that of the Eimeriidea, and takes place either in the intestine, the usual habitat of coccidia, or in endothelial cells of the blood-vessels. The sporozoites which are eventually produced enter the circulating cells of the blood, and are passively transferred by some blood-sucking invertebrate to another vertebrate host. These forms (*Lankesterella* and *Schellackia*) have been considered above (pp. 876 and 878).

In other instances it is the gametocyte which enters the blood-cell, in which case the fertilization process is of the *Adelea* type, and the formation of oöcyst and sporozoites takes place in the blood-sucking invertebrate, which again transfers the sporozoites to another vertebrate. Amongst the hæmogregarines there occur various stages in transition from the typical coccidia to the hæmogregarines, which Reichenow terms hæmococcidia. It is those hæmogregarines which resemble *Adelea ovata* in the behaviour of the macro- and micro-gametocytes that are included in the sub-order Hæmogregarinidea, with the three following families:

1. *Family:* HÆMOGREGARINIDÆ Neveu-Lemaire, 1901. — The small oöcyst produces sporozoites without the formation of sporocysts. The schizogony cycle occurs in the red blood-corpuscles or other cells of the body of vertebrates. After several generations merozoites enter red blood-corpuscles and become micro- or macro-gametocytes. They are then taken up with the blood by an invertebrate (leech), in the gut of which association of the micro- and macro-gametocytes occurs. A small

number (two to four) microgametes is produced, and after fertilization of the adjacent macrogamete the oöcyst is formed.

2. *Family:* HEPATOZOIDÆ.—The large oöcysts contain many sporocysts, which produce numerous sporozoites. The schizogony cycle takes place in cells of the internal organs (liver, spleen, bone marrow, etc.) of vertebrates. After several generations of merozoites have been produced some of them enter red blood-corpuscles or leucocytes, where they become micro- and macro-gametocytes. They are then taken into the body of an invertebrate (tick, mite, etc.), where association of micro- and macro-gametocytes occur. There is some uncertainty as to how fertilization occurs, but the process is probably like that in the family Hæmogregarinidæ. The zygote becomes encysted in the oöcyst, which increases enormously in size, eventually producing sporoblasts, sporocysts, and sporozoites.

3. *Family:* KARYOLYSIDÆ.—The oöcyst formed in the epithelial cells of the intestine of a mite produces a number of sporoblasts, which are liberated from the oöcyst as motile vermicules (sporokinetes), which eventually settle down in the egg of the mite, where they secrete sporocysts, within which are developed a number of sporozoites. The mite hatched from the egg has the sporocysts in its intestinal epithelium, and these are cast off and voided in the fæces, which are eaten by the vertebrate host, a lizard. The sporozoites make their way to the endothelial cells of the vessels, where the schizogony cycle takes place. Eventually, certain merozoites enter the red blood-corpuscles as gametocytes, where they appear as hæmogregarines. The gametocytes are taken up by the mite, in the gut of which syzygy takes place and the oöcyst is formed.

SYSTEMATIC DESCRIPTION OF THE FAMILIES OF THE SUB-ORDER HÆMOGREGARINIDEA.

1. *Family:* HÆMOGREGARINIDÆ Neveu-Lemaire, 1901.

This family contains the single genus *Hæmogregarina* Danilewsky, 1885, which has the characters of the family. The genus was founded by Danilewsky for the hæmogregarine of the tortoise, which was named *H. stepanowi.*

Hæmogregarina stepanowi Danilewsky, 1885.—This is a parasite of the European water tortoise, *Emys orbicularis* (*Cistudo europea*), and the leech, *Placobdella catenigera* (Fig. 452). The life-history was described by Reichenow (1910), to whose investigations much of our knowledge of the hæmogregarines is due (Fig. 451). The tortoise is infected in the first place by sporozoites introduced by a leech. They make their way to the blood-
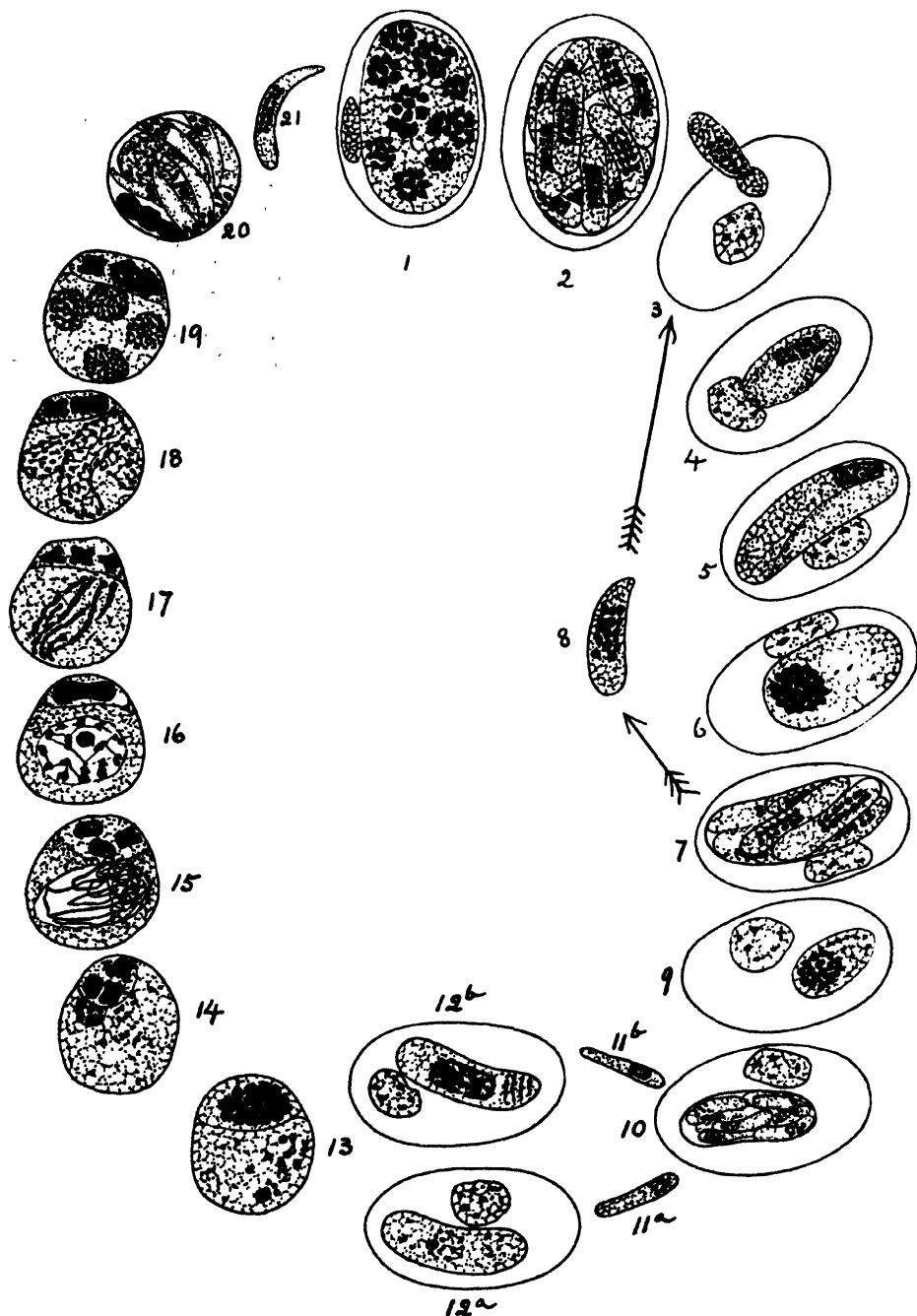
FIG. 451.—LIFE-CYCLE OF *Hæmogregarina stepanowi* (× ca. 1,800). (AFTER REICHENOW, 1910.)

[*For description see opposite page.*

vessels, where they invade the red blood-corpuscles (Fig. 451, 3). Within these they grow into elongate vermicules, which are eventually doubled upon themselves in the form of a U. The two limbs of the loop gradually fuse to form a more solid ovoid body. The various stages of growth from the sporozoite can be seen in blood-films. The fully-grown form is the schizont, and the red cell containing it becomes lodged in the bone marrow. Nuclear multiplication takes place till thirteen to twenty-four nuclei are present, and segmentation into a corresponding number of merozoites ensues (Fig. 451, 4-8). The merozoites are liberated into the blood-stream, and infect new red blood-corpuscles. The number of merozoites produced by a schizont is greater in the earlier stages of infection than in the later. Finally, merozoites develop into much smaller schizonts, each of which produces only six small daughter forms, which are young gametocytes (Fig. 451, 9-10). These enter red blood-corpuscles and grow into gametocytes, which are elongate and slightly curved sausage-shaped bodies. The macrogametocyte (Fig. 451, 11a-12a) consists of denser cytoplasm, and contains a smaller nucleus than the microgametocyte (Fig. 451, 11b-12b). As seen in the red blood-corpuscles they are of approximately equal size, but their further development takes place in the leech (Fig. 452). In the blood of the tortoise, therefore, there occur the young and growing schizonts and gametocytes. When the leech ingests the blood of the tortoise, only the gametocytes develop further, all other stages being destroyed. The gametocytes escape from their host cells, become more or less spherical, and associate in pairs, becoming at the same time adherent to the surface of the intestinal epithelium. A cyst wall is formed round both the macrogametocyte and microgametocyte. The former increases in size, while the latter undergoes little change, except that its nucleus divides to form four separate nuclei, which are the microgamete nuclei. One of the microgametes which are formed fertilizes the macrogamete, a typical fertilization spindle being formed, as in coccidia (Fig. 451, 13-15). The nucleus of the zygote divides to form eight daughter nuclei, after which eight sporozoites and a residual body arise (Fig. 451, 16-21). The oöcyst does not persist, for it soon ruptures and

1-2. Schizogony with large number of merozoites.
3-8. Later schizogony with smaller number of merozoites.
9-10. Final schizogony which gives rise to young gametocytes.
11a-12a. Growth of female gametocyte.   11b-12b. Growth of male gametocyte.
13. Association of male and female gametocytes.
14-15. Male gametocyte producing four male gametes, one of which fertilizes the female, while its nucleus forms a fertilization spindle.
16. Zygote with still attached remains of male gametocyte.
17-19. Nuclear division in zygote.
20. Oöcyst containing eight sporozoites and a residual body. Remains of male gametocyte still present.
21. Sporozoite which enters red cell and initiates the schizogony cycle.

liberates the sporozoites which enter the hæmocœle spaces, and make their way to the dorsal blood-vessel of the leech, whence, by its pulsations, they are driven forwards towards the proboscis. Reichenow thinks they enter the wound in the skin of the tortoise during the feeding of the leech by escaping into the proboscis sheath after actively penetrating the walls of the blood-vessels, or by the rupture of the latter.

There is one point in this cycle which requires explanation, and to which attention was drawn by Minchin (1912). The schizont at first assumes the form of a long looped vermicule, which, according to Reichenow, becomes converted into a solid form by fusion of the two limbs. It is known that *in vitro* these vermicules can be seen to leave the host cell and move about in the plasma. Minchin suggested that this may take place in the blood, and that the vermicule may make its way actively to the bone marrow, where it rounds off as a schizont, either free in the lumen of a small vessel or after entering another cell.

If some such course be not adopted, it is difficult to understand why the vermicule stage occurs at all. The motile vermicule, which is really a partially grown schizont, has been called a *schizokinete* by Minchin and Woodcock (1910).

The life-history of *H. stepanowi*, outlined above, has been confirmed in all essential respects by Robertson (1910) for *H. nicoriæ* Castellani and Willey, 1904, a hæmogregarine of the Ceylon lake tortoise, *Nicoria trijuga*, and the leech, *Ozobranchus shipleyi*. In this case the schizogony producing the large merozoites occurs free in the blood-vessels of the lung, while that producing the smaller gametocytes is found

Fig. 452. — *Placobdella catenigera* (× 2), THE TRANSMITTER OF *Hæmogregarina stepanowi*. (AFTER BRUMPT, 1922.)

in the circulating red blood-corpuscles. After the publication of the results of Reichenow and Robertson the writer made sections of leeches which he had collected from the water tortoise, *Sternothœrus adansonii*, of the Southern Sudan, and which harboured a looped hæmogregarine, which may be *H. stepanowi* (Plate XIX., 16-20, p. 1102). In the intestine of the leech were discovered forms which correspond in every way with those described by Reichenow and Robertson. It seems probable that many other hæmogregarines of cold-blooded aquatic vertebrates will be found to have a similar life-history.

2. *Family:* HEPATOZOIDÆ.

This family, which has been defined above, includes the single genus *Hepatozoon*, founded by Miller (1908) for a parasite of the leucocytes of rats (Plate XIX., 1-7, p. 1102). The first member of the genus to be described was one which occurred in the leucocytes of the dog. It was discovered by Bentley (1905) in India, while James (1905) referred to it as *Leucocytozoon canis*. Later in the same year Balfour (1905) recorded a similar parasite of the red blood-corpuscles of the jerboa (*Jaculus jaculus*) of the Sudan. Laveran (1905c) saw the same parasite in *J. orientalis* of Tunis, and named it *Hæmogregarina balfouri* about a fortnight before Balfour suggested the name *H. jaculi*, which becomes a synonym. Christophers (1905) gave the name *H. gerbilli* to a similar parasite of the red cells of the gerbil (*Gerbillus indicus*) in India. He described the large oöcysts filled with sporocysts in the louse (*Hæmatopinus*). Balfour (1905) first observed the leucocytic hæmogregarine of the rat, *Rattus norvegicus* (*Mus decumanus*) in the Sudan, and in the following year (1906b) proposed to name it *L. muris*. Laveran (1905b) pointed out that both this parasite and the one of the dog ought to be included in the genus *Hæmogregarina*. In an account of the dog parasite, preparations of which were sent him from the Malay States by Gerrard (1906), the writer (1906) came to the same conclusion. Patton (1906) described as *L. funambuli* a similar form in the leucocytes of the Indian palm squirrel (*Funambulus pennantii*). The parasite of *R. norvegicus* was next recorded by Cleland (1906) in Australia, and a form seen by Adie (1906) in *R. rattus* in India was named *L. ratti*. Christophers (1907a) then studied the development of the parasite of the dog in the tick, *Rhipicephalus sanguineus*. He described the association of the parasites in pairs in the stomach of the tick, and the production of cysts containing twelve to fourteen sporozoites. These cysts were actually the sporocysts, the oöcysts having been overlooked. On this account Christophers thought he had possibly been in error in attributing to the parasite of the gerbil the large cysts containing numbers of smaller cysts with sporozoites which he had found in lice. Patton (1908) described the schizogonic cycle of the parasite of the palm squirrel as occurring in the lungs, and recorded two new species: *L. felis domestici* from the cat, and *L. leporis* from the hare, *Lepus nigricollis*. Miller (1908) gave a very full account of the life-history of the parasite of the rat, which he called *Hepatozoon perniciosum*, both in the rat and in the mite, *Lælaps echidninus*.

As regards the correct name for these hæmogregarines of the leucocytes and red blood-corpuscles of mammals, a large number of species of which has been described, it is evident they do not belong to the genus *Leuco-*

cytozoon (p. 903). The life-history of the parasite of the rat as described by Miller (1908), and that of the dog as worked out by Christophers (1907a) and the writer (1911a), together with the observations of Christophers (1905) on the parasite of the red blood-corpuscles of the gerbil and its development in the louse, show that in the production of the large oöcysts containing many sporocysts these parasites differ from those of the genus *Hæmogregarina*. Miller's name *Hepatozoon* becomes, therefore, the correct generic title for these parasites. The name *Leucocytogregarina*, which is employed by some writers, was not used in a generic sense till Sangiorgi (1912) referred to the form in the mouse as *Leucocytogregarina musculi*.

Aragão (1911) described a number of hæmogregarines from South American birds. It has been assumed by Nöller (1920) and others that he was really dealing with toxoplasmata (see p. 1042). The discovery by Adie of an undoubted hæmogregarine in an Indian eagle led Hoare (1924a) to study Aragão's paper. It was found that Aragão was probably dealing with two distinct types of parasite—toxoplasmata in two birds and hæmogregarines in five. The parasite of the Indian eagle, which was named *Hepatozoon adiei*, closely resembles the leucocytic parasites of the dog, rat, and other animals, and it is probable that in five of his birds Aragão was dealing with a similar organism.

Hepatozoon muris (Balfour, 1905).—This parasite was first seen by Balfour (1905) in the brown rat, *Rattus norvegicus*, in the Sudan (Plate XIX., 1-2, p. 1102). Since then it has been recorded from various parts of the world, and will probably be found wherever the common rat has made a home. The same parasite was described from the black rat, *R. rattus*, by Adie (1906), who named it *Leucocytozoon ratti*. It is undoubtedly identical with *H. muris* and *H. perniciosum* studied by Miller (1908) in the white rat. The same parasite was seen by Cleland (1906) and Johnston (1909) in Australia, by Carini (1910) in Brazil, by França and Pinto (1911) in Portugal, by Darling (1912a) in Panama, and by Coles (1914) in England. Kusama, Kasai and Kobayashi (1919) observed it in *R. alexandrinus*, *R. norvegicus*, and *R. rattus* in Japan, and proposed to name it *Leucocytogregarina innoxia*. The most complete account of the life-history of *H. muris* is that of Miller (1908), who had the opportunity of examining a batch of very insanitarily kept white rats which were heavily infested with mites. Massive infection with *H. muris* occurred, many of the animals succumbing to the marked degenerative changes which were produced in the liver. Miller suggested for the parasite the name *H. perniciosum* on account of this pathogenic action, but the organism is undoubtedly the same as that of wild rats, in which such heavy infections do not occur, and which are apparently

little affected by the presence of the parasite. *H. muris* is frequently encountered in small numbers in ordinary blood-films of wild rats. Thus, out of a series of 278 wild rats examined at the Wellcome Bureau of Scientific Research, thirty-two were found to harbour the parasite. In Miller's series of white rats heavily infested with mites all were infected.

The schizogony cycle takes place in the liver, where it can be studied in sections of this organ (Fig. 453, 4-8). Miller was unable to find these stages in any other tissue of the body. The smallest schizont is a spherical uninucleate body about 10 microns in diameter. According to Miller it occurs in the glandular cells, but it is possible that they are in reality endothelial cells of the blood-vessels or the wandering Kupfer cells of endothelial origin. The schizont increases in size, while the nuclei multiply by repeated divisions till twelve to twenty are present. The fully-grown schizont measures on an average 30 by 25 microns, but may reach 35 by 28 microns. It is surrounded by a delicate cyst wall. The nuclei of the schizont arrange themselves on the surface of the two extremities. Small elevations of cytoplasm occur near each nucleus, and finally there are budded off a number of merozoites, leaving a large residual mass of cytoplasm. If a scraping from a liver is examined on the warm stage, the merozoites within the cysts can be seen to be gliding about over one another like a mass of worms. When the cysts rupture, the merozoites enter other cells and repeat the process of schizogony. According to the observations of Kusama, Kasai, and Kobayashi (1919), who studied *H. muris* in Japan, the first schizogony cycle starting from the sporozoite occupies four days. On the fifth day after infection most of the liver cysts are found to be empty, having liberated their merozoites. The growth of these into mature schizonts again occupies four days. A similar cycle is repeated a third time, but after this, the third schizogony, the merozoites, or rather, young gametocytes, instead of entering liver cells, invade the mononuclear leucocytes, when they appear in the blood as typical hæmogregarines. Occasionally a fourth and even a fifth schizogony will occur in the liver. The cyst which is formed around the schizont appears to be a definite structure formed by the parasite rather than the mere thinned-out remains of the host cell. When seen in a fresh scraping from the liver, they have the appearance of ovoid cysts with rigid walls.

The gametocytes which enter the leucocytes, according to Miller and the Japanese observers named above, show no sexual dimorphism (Fig. 453, 9-10). They are all of one type, and are enclosed in definite capsules which offer some resistance to the penetration of stains. They undergo no further change till they are ingested by the mite, *Lælaps echidninus*, which sucks the blood of the rat (Fig. 454). During the first twenty-four hours the blood undergoes digestion, and the hæmogregarines
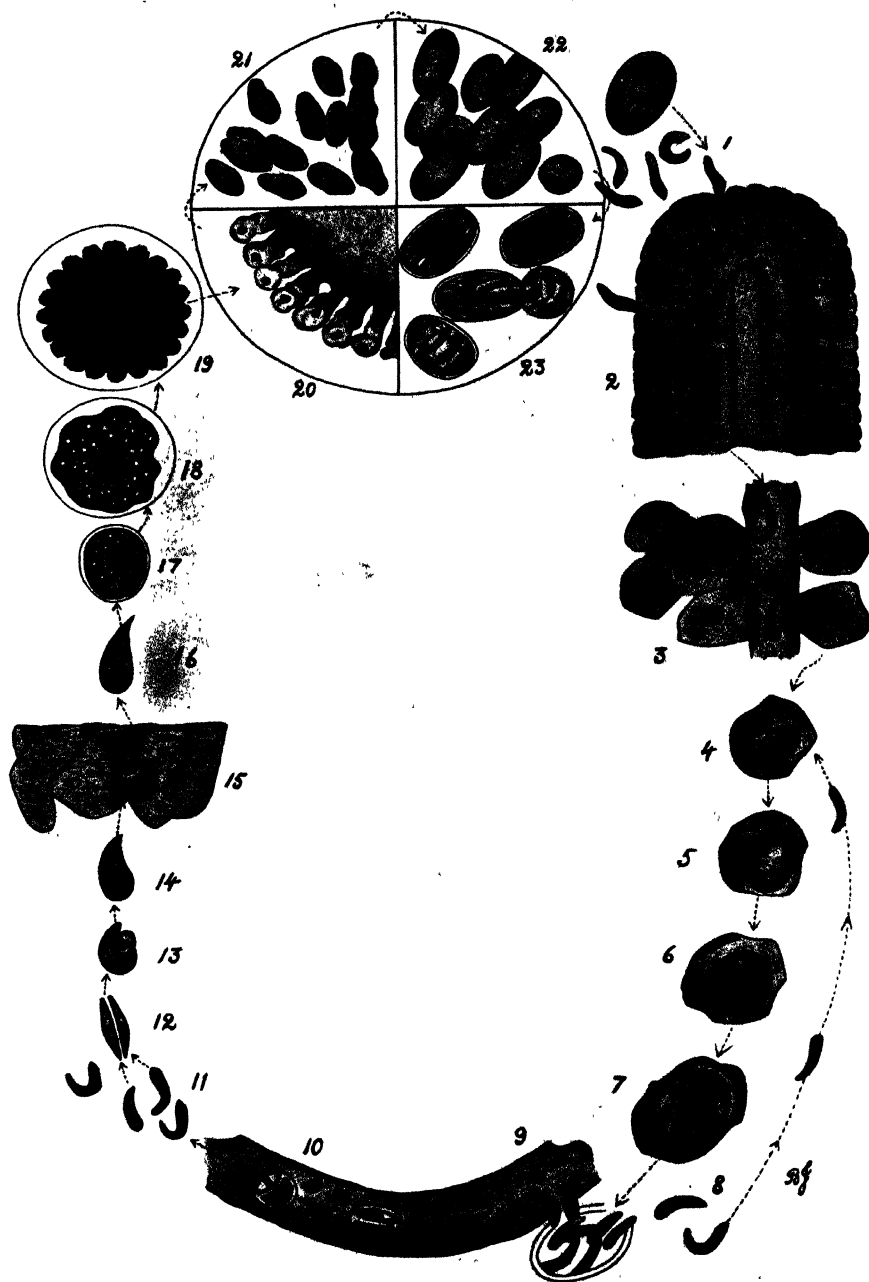
FIG. 453.—LIFE-CYCLE OF *Hepatozoon muris* IN THE RAT AND MITE, *Lælaps echidninus.* (AFTER MILLER, 1908.)

[*For description see opposite page.*

are liberated from the leucocytes and escape from the enclosing cysts. They then associate in pairs, each member of a pair becoming flattened to produce an elongate body with pointed extremities (Fig. 453, 11-12). According to Miller and also the Japanese workers, complete fusion of the two gametocytes takes place, their nuclei also uniting. Before actual union occurs, one, which may be considered the macrogametocyte, increases somewhat in size and encloses the smaller microgametocyte (Fig. 453, 13-14). From what is known of other hæmogregarines, such as *H. stepanowi*, described above, it is probable that the microgametocyte produces microgametes, and that a fertilization of the *Adelea* type occurs instead of the complete fusion of the two gametocytes. This point undoubtedly requires further investigation. After fertilization the zygote elongates and becomes a motile oökinete measuring 25 by 10 microns. It moves about in the stomach contents and grows, till at the end of forty-eight hours after the blood had been taken up by the mite it is full-grown, and measures 25 by 50 microns. In sections of the mite at this period the oökinetes are seen adherent to the surface of the cells, actually between them or in the tissues of the body (Fig. 453, 15-16). It is evident they migrate through the intestinal wall to the surrounding tissues. Here they settle down, become spherical, and commence growing. When the growing zygote is 60 to 75 microns in diameter it has a delicate cyst wall. Growth is continued till the cyst has a diameter of 100 to 150 microns. This stage is reached about five days after the mite's feed. At this time the nucleus divides into two, and after repeated divisions as many as fifty to one hundred are present. During this nuclear multiplication further growth of the cysts takes place, till a maximum diameter of 200 to 250 microns is reached (Fig. 453, 18-19). The surface of the cytoplasm now becomes covered with numerous bud-like projections which increase in size. Each of these, with a single nucleus, is separated from the residual cytoplasm as a sporoblast which measures 15 by

1. Escape of sporozoites from sporocysts in intestine of rat.
2. Penetration of intestinal epithelium by sporozoites and their entry into the blood-vessels of villi
3. Passage of sporozoites from blood-vessels of liver and penetration of liver cells.
4-7. Growth of schizont and formation of merozoites in liver cells.
8 Escape of merozoites from liver cells and their entry into other cells to repeat schizogony.
9. Merozoites (gametocytes) leaving liver cells to enter blood-vessels.
10. Gametocytes in mononuclear cells of blood.
11. Escape of gametocytes from mononuclear cells in the stomach of the mite.
12-15. Syngamy and penetration of intestinal wall by zygote (oökinete).
16-19. Growth of zygote (sporont) in oöcyst in tissues of mite.
20. Portion of surface of sporont, showing method of sporoblast formation by budding.
21. Portion of oöcyst containing sporoblasts.
22. Portion of oöcyst containing sporoblasts with multiplying nuclei.
23. Portion of oöcyst containing sporocysts, in each of which are a number of sporozoites. The mite is eaten by the rat, in the intestine of which the sporozoites escape from the sporocyst.

10 microns. The nucleus of the sporoblast multiplies by repeated divisions, and a cyst, the sporocyst, is formed around it. Within each sporocyst are formed from twelve to twenty-four sporozoites, which are closely packed around a residual body. In sections of heavily infected mites, groups of sporocysts which have escaped from the ruptured oöcyst may be found scattered all through the tissues (Fig. 453, 20-23).

Miller succeeded in infecting rats by placing on them infected mites (Fig. 454). Rats were also infected by contaminating food with crushed mites. Injection of crushed infected mites into the peritoneal cavity
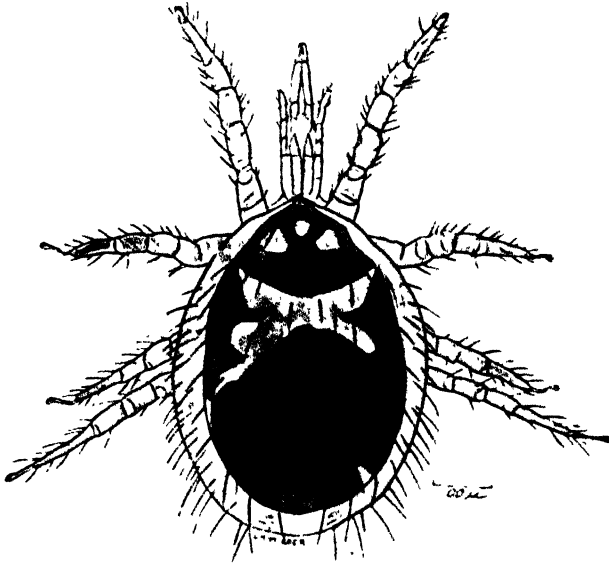
Fig. 454.—*Lælaps echidninus* (♀), THE TRANSMITTER OF *Hepatozoon muris* (× 8). (AFTER MILLER, 1908.)

did not produce infection. It is thus evident that infection of the rat takes place by way of the intestinal canal, and is probably brought about by the rats devouring the infected mites. It was noted that the fluid from the duodenum of the rat had a stimulating effect on the sporozoites within the sporocysts. Under its influence the sporozoites commenced moving within the sporocyst, which eventually burst and liberated the sporozoites, which continued their movements on the slide.

It is not improbable that *H. musculi*, described from the mouse by Porter (1908a), Sangiorgi (1912), and Yakimoff and Schokhor (1907a), is actually *H. muris*.

Hepatozoon canis (James, 1905).—This parasite, which was discovered by Bentley (1905) in India, is very similar to *H. muris* (Plate XIX., 3-4, p. 1102). It occurs in the leucocytes of dogs in various parts of the world. James (1905) observed it in India, and gave the name *Leucocytozoon canis* to the parasite. It was discovered by Gerrard (1906) in Malaya, by Christophers (1906) in Madras, by Mathis and Leger (1909) in Tonkin, by Lebœuf and Ringenbach (1910) in the Congo, by Kleine (1910) near Tanganyika, by Yakimoff and Kohl-Yakimoff (1911) in Tunis, by Basile (1911*b*) in Italy, by the Sergents and Senevet (1912) in Algeria, by Leger, A. (1912), in Senegal, and by the writer (1911) in Bagdad. Though the forms in the cat, jackal, and hyæna have been given different names, it is far from clear that they are actually distinct species. In fact, all these parasites resemble one another so closely that it is almost, if not quite, impossible to separate them on morphological grounds alone. Leger, A. (1912*a*), believed that the dogs of Senegal were liable to infection with two species, one of which was referred to as *Hæmogregarina canis* and the other as *H. chattoni*, a name which he had previously given to the parasite of the hyæna. Martoglio (1913) believed that the parasite of dogs in Abyssinia differed from the common *Hepatozoon canis*, and resembled the one seen by Patton (1910) in the jackal, and which he had named *Hæmogregarina rotundata*. Martoglio named the form in the Abyssinian dog *H. rotundata canis familiaris*, and referred to Patton's parasite as *H. rotundata canis aurei*, and to Leger's parasite (*H. chattoni*, of the African hyæna) as *H. rotundata hyenæ crocutæ*. This trinomial terminology is not in accord with the rules of nomenclature.

From the work of Christophers (1906, 1907) and of the writer (1911), it appears that the schizogony cycle of *Hepatozoon canis* takes place in the spleen and bone marrow. Rau (1925) has seen schizonts in the liver also. The gametocytes, which occur in the leucocytes, undergo development in the common dog tick, *Rhipicephalus sanguineus* (Fig. 420), in which, as in the case of *H. muris* in the mite, there are eventually produced large oöcysts about 100 microns in longest diameter, containing from thirty to fifty sporocysts about 15 to 16 microns in length, each of which includes about sixteen sporozoites and a residual body (Fig. 455). The sporozoites are 14 to 15 microns in length. After the sporocysts are fully developed the oöcyst appears to break up, and sporocysts are found scattered about amongst the tissues of the tick. Infection of the dog is probably produced by the ticks being eaten. In the case of *H. canis* the schizonts, which occur in the spleen, liver, and bone marrow, are of several types (Fig. 39). At one end of the scale there are schizonts which produce a small number of large merozoites, while at the other there are schizonts producing a large number of small ones. The small merozoites are un·

doubtedly the forms which enter the leucocytes, while the large ones are probably destined to become schizonts again. Between the two extremes there are connecting forms which produce merozoites of intermediate size. If the spleen pulp of an infected dog is broken up in saline solution and the emulsion examined in wet preparations, the schizonts or groups of merozoites are seen enclosed in a definite cyst. The wall appears to be of the thickness of a cyst of *Entamœba coli*. Whether this
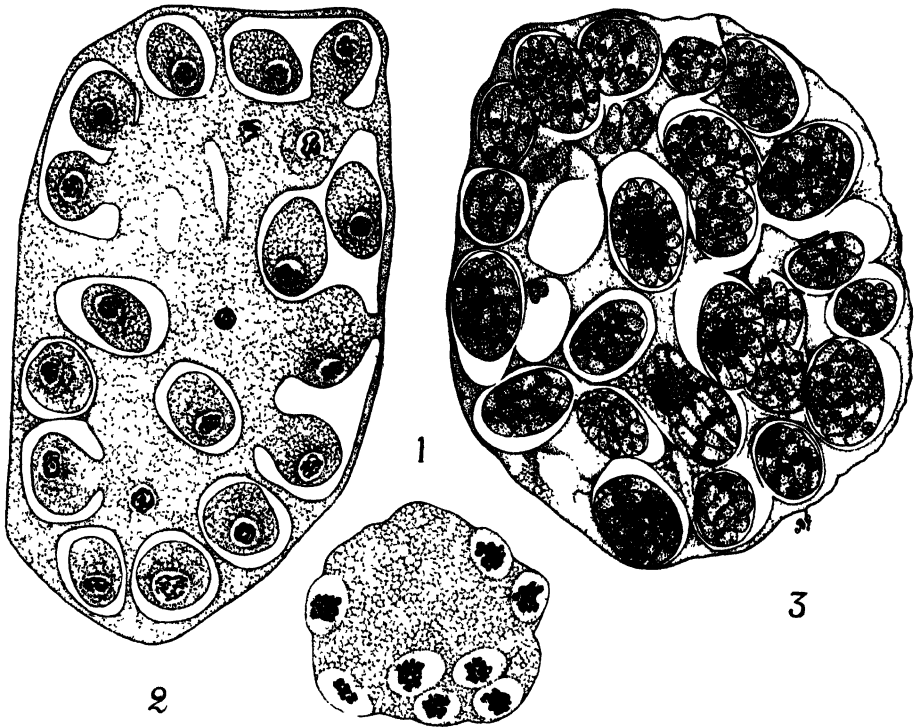
FIG. 455.—*Hepatozoon canis:* DEVELOPMENTAL STAGES IN THE TISSUES OF THE TICK, *Rhipicephalus sanguineus* (× 600). (ORIGINAL.)

1. Zygote (sporont) which has increased considerably in size and developed a number of nuclei.
2. Section of fully-grown sporont in which sporoblast formation is taking place.
3. Section of fully-developed oöcyst in which the sporocysts, each containing about sixteen sporozoites, have formed.

cyst wall is developed from the remains of the host cell or whether it is a special membrane formed by the parasite cannot be stated.

Rau (1925) claims to have infected dogs by injection of spleen material containing schizonts, as also by the injection of the tissues of infected ticks. Parasites appear in the blood of dogs in two or three weeks after inoculation, and it is stated that the infection progresses in intensity and may bring about the death of the animals. It is by no means clear from his account that other causes of death were excluded.

Hepatozoon criceti Nöller, 1912.—This parasite of the hamster (*Cricetus frumentarius*), recorded by Nöller (1912c), has a life-history very similar to that of *H. muris* and *H. canis*. The invertebrate host, according to Nöller (1920b), is the mite *Liponyssus arcuatus*, in which large oöcysts are produced like those described above.

The hæmogregarines which occur in the red blood-corpuscles of mammals develop in a similar manner (Plate XIX., 5-7, p. 1102). Two of the best-known are *Hepatozoon balfouri* (Laveran, 1905) of the jerboas (*Jaculus gordoni, J. orientalis, J. johnstoni*) and *H. gerbilli* (Christophers, 1905) of the gerbil (*Gerbillus indicus*). The invertebrate host of the former is probably a mite (*Dermanyssus* sp.) or a flea (*Pulex cleopatræ*), and of the latter the louse (*Hæmatopinus stephensi*), in which Christophers found large oöcysts containing sporocysts, each of which had six to eight sporozoites.

Large oöcysts of this type were discovered by Chatton and Roubaud (1913) in the body cavity of a *Glossina palpalis*. As these observers suggest, they are probably derived from some unidentified hæmogregarine. Similar cysts were seen by Macfie (1916) in these flies. They were 400 to 560 microns in diameter, and contained many sporocysts, each having a diameter of 36 microns, and containing about forty sporozoites measuring 23 by 4 microns.

Adler and Theodor (1925) have discovered in a single *Phlebotomus papatasii* in Jericho large oöcysts of what may be a species of *Hepatozoon*. The oöcysts measured 130 by 95 microns, and each contained about one hundred sporocysts and a number of round refractile bodies 8 microns in diameter. The sporocysts varied in length from 21·4 to 36·4 microns, and in breadth from 15·7 to 20 microns. Each contained from four to sixteen sporozoites, a residual body from 3·6 to 6·4 microns in diameter, and a number of small refractile granules. Within the sporocyst the sporozoites were actively motile. When the sporocysts were ruptured it was noted that each sporozoite was enclosed in a spindle-shaped membrane measuring 35 by 6·4 microns. On one side of the membrane two longitudinal parallel lines could be seen. These evidently represented the margins of a slit-like opening, for sporozoites were seen to emerge from the membrane between the lines. The sporozoite when fully extended measured about 35 by 5 microns. Apart from the membrane enclosing the sporozoites, which has not been seen in other forms, the parasite corresponds with the similar stages of *H. muris*, *H. canis*, and the cysts discovered by Chatton and Roubaud (1913) in *Glossina palpalis*. It is not improbable that the oöcysts were derived from the blood of some animal harbouring hæmogregarines on which the sand fly had fed.
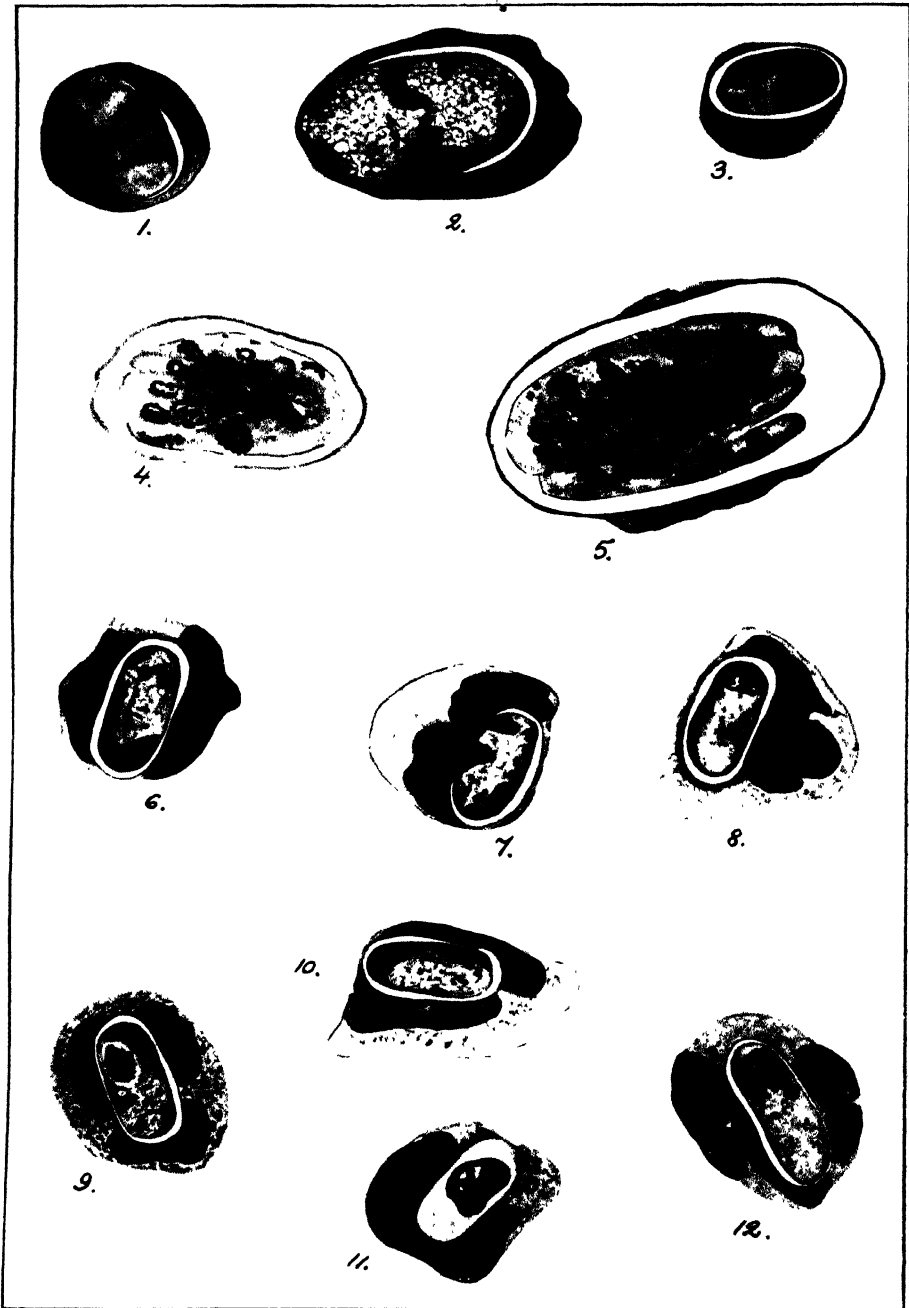
FIG. 456.—*Hepatozoon adiei* OF AN INDIAN EAGLE (× 2,000). (AFTER HOARE, 1924 ; FROM *Trans. Roy. Soc. Trop. Med. and Hyg.*, vol. xviii., p. 64.)

Hæmogregarines similar to the above have been described from a number of mammalian hosts, but in most cases they have been seen only in the blood stage. They will be referred to below (p. 1111).

Hepatozoon adiei Hoare, 1924.—This is a parasite of an Indian eagle, the correct name of which could not be obtained. The parasite so closely resembles *H. muris* and *H. canis* that a separate description is not necessary (Fig. 456). The schizonts were found in smears of the lung, and they appeared to resemble those of the dog and rat parasite. Only dried films of the blood and organs of the bird were available for study, and nothing is known of the invertebrate cycle.

Fantham (1924) describes as *Leucocytogregarina amadinæ* a hepatozoon of *Amadina erythrocephala*. Assuming that the parasites which Aragão (1911) discovered in five South American birds belonged to the same genus, the following additional species can be recognized: *H. atticoræ* (*Atticora cyanoleuca*), *H. rhamphocæli* (*Rhamphocœlus brasilius*), *H. paroariæ* (*Paroaria larvata*), *H. tanagræ* (*Tanagra palmarum*), *H. brachyspizæ* (*Brachyspiza capensis*) (see p. 1086).

3. *Family:* KARYOLYSIDÆ.

The definition of this family has been given above (p. 1081). It contains the single genus *Karyolysus* (*Caryolysus*), which was found by Labbé (1894) for a parasite which had been discovered in the red blood-corpuscles of lizards of the genus *Lacerta* by Danilewsky (1886), who named it *Hæmogregarina lacertarum*. Owing to the fact that the parasite often has a karyolytic action on the nucleus of the host cell and causes it to fragment, Labbé placed it in a new genus, *Karyolysus*. From the work of Reichenow it appears that the parasite *K. lacertarum* differs in its life-cycle from other hæmogregarines, and that it correctly belongs to a distinct genus for which Labbé's name *Karyolysus* can be employed, though the characteristic feature of the genus given by Labbé— namely, the karyolytic action—cannot be regarded as of generic value.

Karyolysus lacertarum (Danilewsky, 1886).– The life-history of this hæmogregarine, which is a parasite of the lizard, *Lacerta muralis*, has been described by Reichenow (1921). The schizogony stages occur in the endothelial cells of the capillaries, where they may be detected in sections of any of the organs of the body (Fig. 457, 1-4). The youngest schizont is derived from a sporozoite or a merozoite. It has an elongate form, but by growth gradually becomes more ovoid, while its cytoplasm becomes charged with granules of reserve food material. The schizont then forms

1-5. Schizogony stages as seen in dried smears of the lung.
6-10. Forms (gametocytes) in the leucocytes of the peripheral blood.
11-12. *H. muris* and *H. canis* for comparison.
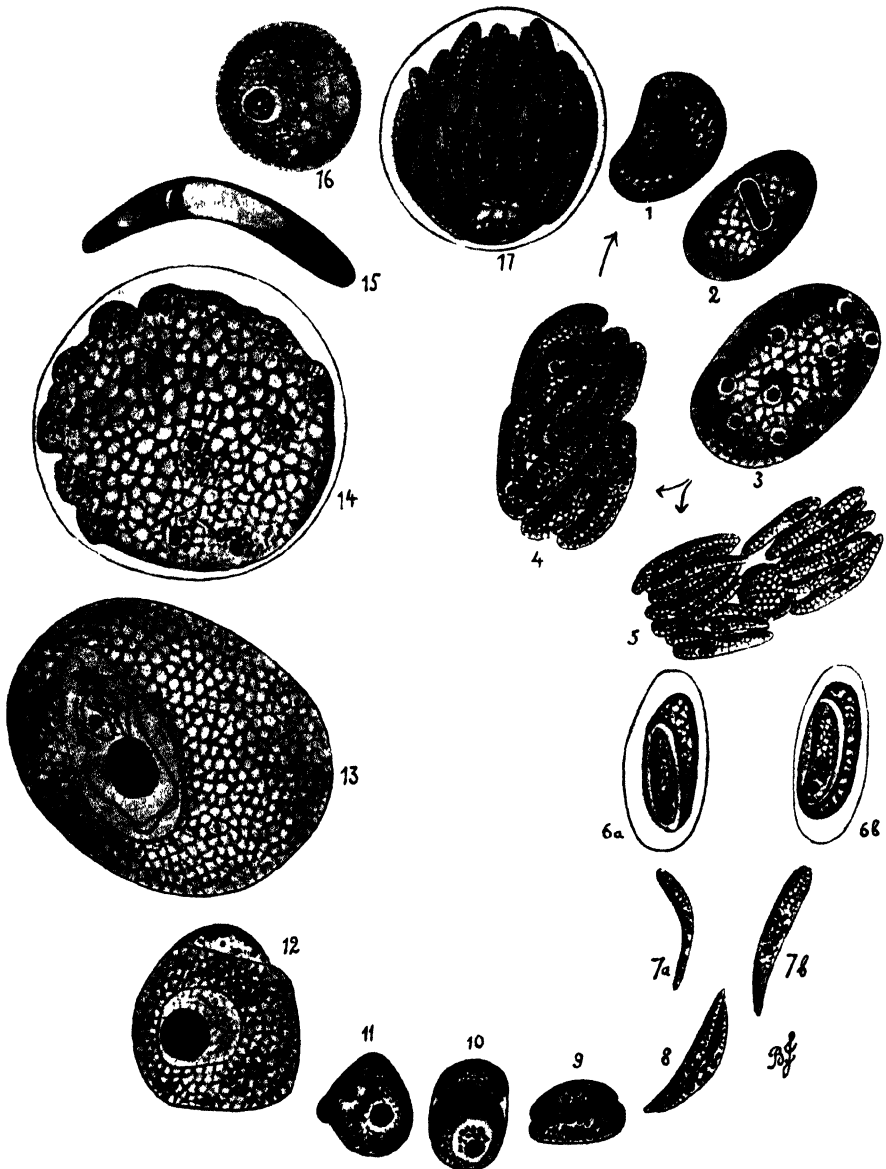
FIG. 457.—LIFE-CYCLE OF *Karyolysus lacertarum* IN THE LIZARD, *Lacerta muralis*, AND THE MITE, *Liponyssus saurarum* (× 1,000).   (AFTER REICHENOW, 1921.)

1-4. Schizogony in the endothelial cells of the blood-vessels of inner organs of the lizard.
5. Schizogony giving rise to smaller merozoites which enter red blood-corpuscles.
6. Merozoites, now male (6a) and female (6b) gametocytes, in red blood-corpuscles.
7. Free male and female gametocytes in intestine of mite.
8. Association of male and female gametocytes.

around itself a membrane, after which further growth takes place, while the nucleus multiplies by repeated divisions. At all stages the nucleus consists of a membrane enclosing a group of chromatin granules and a karyosome (binnenkörper), which stains less deeply than the chromatin and has a lateral position in the nucleus. At each nuclear division the karyosome divides by dumb-bell constriction, and the chromatin granules are also divided. Eventually, eight to thirty nuclei are produced, and by segmentation of the schizont a corresponding number of merozoites is formed, together with a residual body. The merozoites may remain for a considerable time within the cyst, during which they increase in size by absorbing nutriment from the residual body, which may ultimately disappear entirely. There is considerable variation in the size and details of structure of the merozoites from different cysts. Finally, the cyst ruptures, the merozoites escape into the blood-stream and invade other endothelial cells, where the schizogony is repeated.

After schizogony has been repeated a number of times, the schizonts give rise to a larger number of small merozoites which invade the red blood-corpuscles. In structure these do not differ from other merozoites except that the karyosome cannot be distinguished in the nucleus (Fig. 457, 5). They are in reality the young gametocytes, and soon after their entry into the red blood-corpuscles they become surrounded by a fairly tough membrane. The nucleus of the red cell is indented and pushed to one side, where it not infrequently breaks up. The form which will become the macrogametocyte grows slightly, and a karyosome becomes apparent at one end of the nucleus (Fig. 457, 6b-7b). Those which become micro-gametocytes remain unchanged (Fig. 457, 6a-7a). On account of this growth it results that in any blood-film various types of hæmogregarine occur. The further development takes place in the female mite, *Liponyssus saurarum* (Fig. 458). The micro- and macro-gametocytes become free in the gut, and can be distinguished from one another (Fig. 457, 7a-7b). The macrogametocyte is larger, has a larger nucleus, and a more distinct karyosome than the microgametocyte, which has developed a karyosome much later. While still free in the gut the micro- and macro-gametocytes become associated in pairs and flattened against one another to form

9. Growth of male and female gametocytes.
10. Female gametocyte is still large, while the male gametocyte is producing two microgametes.
11. One microgamete has entered the female gamete.
12. Growth of zygote; the remains of the male gametocyte are still present.
13. First nuclear division in the zygote (sporont).
14. Multinucleated sporont about to produce sporoblasts.
15. A free motile sporoblast (sporokinete) which makes its way to the eggs of the mite.
16. Rounded off sporokinete in the egg of the mite.
17. The rounded-off sporokinete becomes encysted (sporocyst), and appears in the tissues of the newly-hatched larva, and becomes mature with twenty to thirty sporozoites in the nymph. The nymph is devoured by the lizard, in the intestine of which the sporozoites escape and make their way to the endothelial cells of the blood-vessels.

elongate spindle-like bodies. In this condition each pair enters an epithelial cell of the gut, where it becomes more or less rounded and enclosed in a cyst (Fig. 457, 8-9). The macrogametocyte increases still further in size, whereas little growth of the microgametocyte takes place. The nucleus and the karyosome of the macrogametocyte become larger, while that of the microgametocyte divides once to form two nuclei, which are separated off as two flagellated microgametes, one of which fertilizes the macrogamete (Fig. 457, 10-11). In this process a typical fertilization spindle occurs. After the chromatin from the microgamete nucleus has entered the spindle, the latter retracts and becomes a spherical nucleus.

The zygote now increases enormously in size. Nuclear multiplication commences, and at each division the karyosome divides, as in the case of the schizonts. The first division of the synkarion is a reduction division (Fig. 457, 13). The number of its chromosomes is double the usual number, owing to the entry of the chromo-somes of the microgamete. Instead of dividing as they do at other stages, the chromosomes, which, as regards size, are arranged as pairs of homologous chromo-somes, separate into two groups, each of which enters a daughter nucleus. One chromosome of each pair enters into each group. At the second and subsequent divisions the number of chromosomes is maintained by actual splitting of each chromosome to form daughter chromo-somes. After nuclear multiplication is completed, division into a varying number
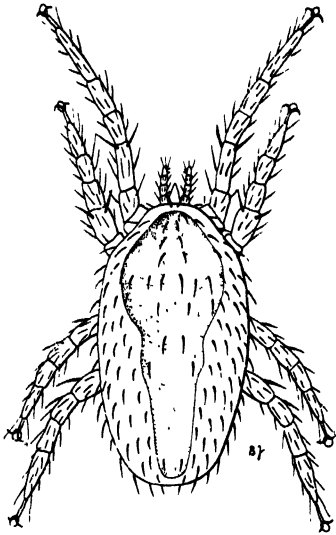
FIG. 458.—*Liponyssus saurarum* (♀), THE TRANSMITTER OF *Karyolysus lacertarum* (× ca. 50). (AFTER OUDEMANS, 1902, SLIGHTLY MODIFIED.)

of elongate vermiform bodies occurs (Fig. 457, 14-15). They reach a length of 40 to 50 microns, and are characterized by having homogeneous aggregations of food reserve material in the cytoplasm. A residual body is generally present. The vermiform sporoblasts are motile, and except that they are larger, resemble the oökinetes of other parasites. To distinguish it from an oökinete or motile zygote, the motile sporoblast has been called *sporokinete* by Reichenow (Fig. 457, 15). After rupture of the oöcyst, the sporokinetes make their way into the body cavity of the mite and wander about amongst its various tissues. Some of them reach the ovaries, where they enter the ova, become rounded off, and enclosed in

sporocysts. Within each sporocyst nuclear multiplication takes place till twenty to thirty nuclei are present. Division into a corresponding number of sporozoites and a residual body follows (Fig. 457, 16-17). While nuclear multiplication is in progress the larva hatches from the egg, and by the time it becomes a nymph the sporocysts are fully formed. The mature sporocysts measure from 20 to 25 microns in diameter.

The nucleus of the synkarion has a distinct nuclear membrane and karyosome. During its division the membrane and karyosome persist, the latter dividing at each nuclear division, so that the nucleus of the sporokinete has a similar structure. At the first division of the sporoblast nucleus within the sporocyst the nuclear membrane is still present, but it becomes less distinct. When the two daughter nuclei divide the membrane is no longer seen, there being merely a group of granular thread-like chromosomes, amongst which the karyosome can still be distinguished. At each division each chromosome divides, as also does the karyosome. The latter, however, becomes smaller, so that when the sporozoites are eventually formed each has a nucleus consisting of a few chromatin granules, amongst which a karyosome may or may not be recognized.

During the differentiation of the various tissues of the embryo within the egg the sporocysts occupy cells which are destined to become the lining epithelium of the gut, so that by the time the nymph has its first feed of blood the epithelial cells of the intestine have mature sporocysts within them. After the feed many of the cells are shed into the lumen of the intestine, and the sporocysts with them pass out of the body in the fæces. These are ingested by lizards, which become infected. Infection may also be brought about by the lizard eating the nymph. The sporozoites make their way through the gut epithelium of the lizard to the blood-vessels, into the endothelial cells of which they penetrate, to commence the schizogony cycle.

The account of the life-history of *Karyolysus lacertarum* just given was the outcome of investigations conducted by Reichenow in Italy and Madrid. França had described as many as fourteen species of hæmogregarine from the lizards which occur in the vicinity of Lisbon. Woodcock (1912) expressed the view that all the forms occurring in two of these (*Lacerta muralis* and *L. ocellata*) would be found to belong to one species. Reichenow's investigations (1921) of the complete cycles has shown, however, that the wall lizard, *L. muralis*, from the neighbourhood of Madrid, harbours no less than eight distinct species of hæmogregarine (blood coccidia), which are different from the one studied by him in this lizard in Italy. All but two of these resemble *K. lacertarum* in their cycles of development. The two forms which do not agree with *K. lacertarum* belong to Reichenow's

genus *Schellackia*, which has been already considered, and in which the intra-
corpuscular blood forms are sporozoites and not gametocytes (Fig. 379).

The forms which resemble *K. lacertarum* in life-history fall into two
groups. In one the oöcyst forms in the gut epithelium of the mite, while
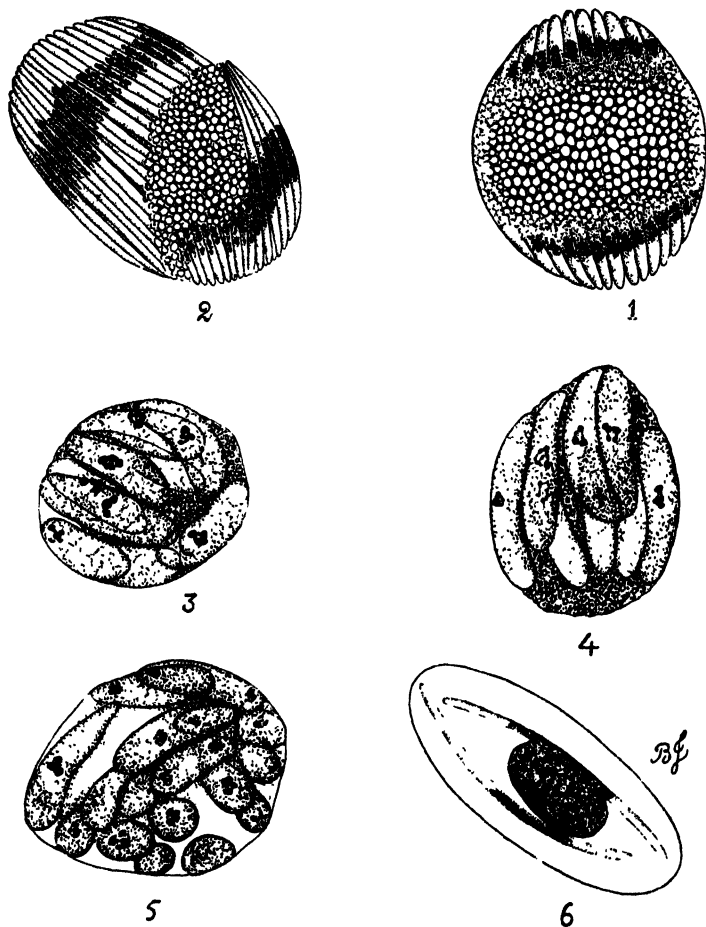
FIG. 459.—SCHIZOGONY OF *Hæmogregarina* (? *Karyolysus*) *gracilis* IN LIVER OF
THE LIZARD, *Mabuia quinquetæniata* OF THE SUDAN (× 3,000). (ORIGINAL.)

1. Commencing production of narrow merozoites (gametocytes) as finger-like outgrowths from
   ends of schizont.   2. Narrow merozoites and large residual body.
3-4. Schizogony into eight large merozoites.   5. Schizogony into about sixteen merozoites.
   6. Red blood corpuscle of peripheral blood containing two of the narrow merozoites
   (gametocytes).

in the other it is formed in the body cavity. To the former group belong
K. lacertarum and *K. bicapsulatus* of *Lacerta muralis*, while to the latter
belongs *K. biretortus* of *L. viridis*.

Undoubtedly many hæmogregarines, especially those of lizards and snakes, will be found to belong to the genera *Karyolysus* and *Schellackia*, but till their life-cycles are known it will be impossible to classify them with any certainty. It is possible, as Reichenow has suggested, that the form described by the writer (1909) as *Hæmogregarina gracilis* from the lizard, *Mabuia quinquetæniata*, of the Southern Sudan belongs to this genus (Plate XIX., 14-15, p. 1102). The complete life-history is not known, but the schizogony cycle with the production of large merozoites occurs in the liver (Fig. 459, 3-5). Finally, a different type of schizogony takes place in that there is produced a large number of very narrow daughter individuals with elongated nuclei (Fig. 459, 1-2). It is these forms which appear in the red blood-corpuscles as hæmogregarines (Fig. 459, 6). They are probably the gametocytes, and are of interest on account of their slenderness, in which respect they differ from the more usual type of hæmogregarine.

HÆMOGREGARINES IN GENERAL.

The hæmogregarines have already been considered above, but it will be convenient to discuss the group as a whole. The vast majority have only been seen in blood-films, where they may be sporozoites, gametocytes in various stages of growth, or schizonts, which, though usually in the uninucleate condition, may sometimes be multinucleate, or actually in process of breaking up into merozoites (Plate XIX., p. 1102). Generally, however, the multinucleate stages of the schizont are confined to the internal organs.

When blood containing intracellular hæmogregarines is mixed with citrate or saline solution, or simply observed in a wet film preparation, the parasites may be seen to leave the host cell, when they move about as elongate vermicules which have the anterior end blunter than the posterior. They often leave the cells in the blood-vessels of animals which have been dead for some hours. It is possible that in the circulating blood the vermicular forms may be able to leave one cell and enter another. Stebbins (1905) and Dobell (1910), as also did Labbé (1894), stated that they had actually observed the free hæmogregarines entering other red blood-corpuscles. It may be that this behaviour of the parasite is the result of exposure on the slide in the same way that flagellation of malarial parasites takes place after removal of blood from the body, but Minchin (1912) considered that if such migrations did not occur in the vertebrate, it would be difficult to account for the fact that the vermicular form within the red cell was retained.

Within the cell in dry films stained by Romanowsky stains the hæmogregarines have a blue-staining cytoplasm, and a more or less centrally

placed purple red nucleus. The latter is a comparatively large structure, and takes the colour of the nuclei of leucocytes rather than the brighter red of the nuclei of malarial parasites. In wet fixed films the nucleus is found to consist of a nuclear membrane upon which granules of chromatin are arranged. There may be a karyosome within the nucleus.

Some hæmogregarines are much shorter than the host cell, and they lie in the stroma of the cell as elongate vermicules, as in the case of *Lankesterella minima*. Others are much longer than the cell, and are then looped in the form of a **U**, the two limbs lying side by side, as in *Hæmogregarina stepanowi* (Fig. 451, 5). In other cases they appear as more solid bodies, about two or three times as long as they are broad, and with rounded ends. These forms appear to be developed from smaller ones which have grown to this shape, or from the looped forms by fusion of the two limbs, or by gradual withdrawal of one limb.

The hæmogregarines within the cells are generally enclosed in a definite cyst, which may be formed by the parasite or from the stroma of the cell. The cyst wall is quite colourless and transparent, and, though usually readily permeable, in some cases offers considerable resistance to the penetration of stains. When the hæmogregarine leaves the cell the cyst wall ruptures, and it glides through the opening. Sometimes, through breaking-down of the host cell, the cysts still enclosing the parasite may be free in the plasma. The remains of cysts may sometimes be seen in films as red-staining, rod-like structures formed by the rolling up of the empty cysts on their longitudinal axis. In blood-films made from animals which have died, empty cysts distorted and folded in various ways are often seen. In certain hæmogregarines of snakes, Sambon and Seligmann (1907) have figured a line across the cyst at each end, and they have supposed that the ends of the cysts are valves which open to liberate the hæmogregarine. This hardly seems probable, for, when the parasites are observed to leave the cyst, they do so through an irregular tear, there being no indication of the opening of a valve. Similar markings were noted by Laveran and Pettit (1909) in a hæmogregarine of *Pituophis melanoleucus*.

The hæmogregarines of cold-blooded animals which have nucleated red blood-corpuscles are usually in these cells. In any infection, however, occasional parasites may be seen within the leucocytes. In some cases, however, these animals appear to harbour hæmogregarines which are definitely leucocytic. Thus Leger, M., and Mouzels (1917b) described such a form from the lizard, *Tupinambis nigropunctatus*, while in the same year Yakimoff described as *H. ninæ kohl-yakimovi* a leucocytic hæmogregarine of an unnamed fish of Transcaucasia. In the case of mammals certain hæmogregarines infect the red blood-corpuscles, while others are

PLATE XIX.

VARIOUS HÆMOGREGARINES IN THE RED BLOOD-CORPUSCLES OF VERTEBRATES. DRIED BLOOD-FILMS STAINED WITH ROMANOWSKY STAINS. (× 2,000).

1-2. *Hepatozoon muris* of the rat.
3-4. *Hepatozoon canis* of the dog.
5-7. *Hepatozoon balfouri* of the jerboa.
8-10. *Hæmogregarina* (?) *najæ* of the African cobra, *Naja hajæ*.
11-13. *Hæmogregarina* (?) *ranarum* of the frog.
14-15. *Karyolysus gracilis* of the lizard, *Mabuia quinquetæniata*.
16-20. *Hæmogregarina stepanowi* (?) of the tortoise, *Sternothærus adansonii*, of the Sudan.
21-22. *Hæmogregarina* (?) sp. of the alligator, *Alligator mississippiensis*.
23-25. *Hæmogregarina* (?) sp. of the gecko, *Tarentola mauritanica*.
26-30. *Hæmogregarina* (?) *æglefini* of the haddock, *Gadus æglefinus*.

(1-25. ORIGINAL; 26-30, AFTER HENRY, 1913.)

PLATE XIX.

exclusively leucocytic forms. Reichenow (1919), however, has shown that the hæmogregarine *Schellackia bolivari* is a parasite of the red blood-corpuscles in the lizard, *Acanthodactylus vulgaris*, and of the lymphocytes in *Psammodromus hispanicus* (see p. 878).

As regards the occurrence of schizonts in the multinuclear or segmenting condition in the peripheral blood, it must be remembered that when this process takes place in the red blood-corpuscles the cells containing the growing schizonts are generally held up in the vessels of the internal organs before nuclear multiplication commences. Sometimes, however, the cells containing the schizonts may appear in the peripheral blood, as occasionally occurs in the case of infection with *Plasmodium falciparum*. Similarly, when the schizogony occurs in the endothelial cells of the vessels, these cells may become dislodged and appear in the peripheral circulation. Labbé (1894) figures stages of schizogony of *Lankesterella* of frogs as occurring in the leucocytes or red blood-corpuscles of the peripheral blood. Nöller (1913b) describes the schizogony of this parasite as occurring in the endothelial cells of the vessels, so that the forms stated by Labbé to be within the leucocytes may in reality be in detached endothelial cells (Fig. 380). The majority of the forms figured by him are in cells of this type. Those in the red blood-corpuscles may actually be schizonts in an exceptional situation, or belong to some totally distinct parasite, such as *Dactylosoma ranarum* (Fig. 431).

In hæmogregarines generally the schizogony stages can only be found in smears or sections of the various organs. The schizonts can often be seen in definite cysts in moist preparations of scrapings from the organs. Ordinary dried smears, owing to shrinkage and distortion, give pictures which are much less satisfactory than sections of properly fixed tissues.

It has been shown above that the hæmogregarines, of which complete life-cycles have been elucidated, fall into two groups. In the first of these the fertilization is of the *Eimeria* type. The male gametocyte produces a comparatively large number of microgametes, and is not developed in association with the macrogamete. The forms which occur in the red blood-corpuscles are sporozoites. In the second group the fertilization is of the *Adelea* type, and the male gametocyte which develops in association (syzygy) with the female gametocyte produces only two or four microgametes. The forms which occur in the red blood-corpuscles are gametocytes, schizonts only being found there in the case of those which reproduce by schizogony in these cells. The hæmogregarines may therefore be grouped as follows:

1. Hæmogregarines which have micro- and macro-gametocytes of the *Eimeria* type. Only sporozoites occur in the red blood-corpuscles, and these are carried from one vertebrate to another by the invertebrate

host (leech or mite) without undergoing further development in the invertebrate.

(*a*) Schizogony and sporogony up to the formation of sporozoites occur in the intestinal wall. When the oöcyst ruptures, the sporozoites enter the red blood-corpuscles. Genus *Schellackia* (Fig. 379).

(*b*) Schizogony and sporogony up to the formation of sporozoites occur in the endothelial cells of the blood-vessels. When the oöcysts rupture, the sporozoites enter the red blood-corpuscles. Genus *Lankesterella* (Fig. 380).

2. Hæmogregarines which have micro- and macro-gametocytes of the *Adelea* type. The blood-corpuscles (either red or white) contain gametocytes and sometimes schizonts. The gametocytes are taken up by an invertebrate, in which the oöcyst containing sporozoites is produced. The sporozoites are transferred to the vertebrate through the agency of the invertebrate.

(*a*) Schizogony occurs in the red blood-corpuscles, and in these cells gametocytes are also formed. In the invertebrate (leech) an oöcyst containing free sporozoites is produced. Genus *Hæmogregarina* (Fig. 451).

(*b*) Schizogony takes place in cells of the internal organs, usually the endothelial cells of the blood-vessels. The gametocytes enter either the red blood-corpuscles or the leucocytes, and in the invertebrate (tick, mite, louse) develop into large oöcysts containing a number of sporocysts, each of which contains the sporozoites. Genus *Hepatozoon* (Fig. 453).

(*c*) Schizogony occurs in the endothelial cells of the blood-vessels, and the gametocytes enter the red blood-corpuscles. In the invertebrate (mite) there is developed an oöcyst in which sporoblasts are produced. These escape from the oöcyst as motile vermicules (sporokinetes) and enter the egg, in which they become sporocysts, within which sporozoites are developed. The young hatching from the egg have the sporocysts in their intestinal epithelium, and the sporozoites are transferred to the vertebrate by them. Genus *Karyolysus* (Fig. 457).

It will be evident that if the life-history of all hæmogregarines were known, it might be possible to group them according to the above classification. In the great majority of cases only the blood forms have been described, and as it is not certain whether these are gametocytes, sporozoites, or schizonts, it is impossible to determine the genera to which they belong.

Hæmogregarines of Aquatic Reptiles and Amphibia.

The life-history of *Hæmogregarina stepanowi* of the water tortoise was described by Reichenow (Fig. 451). In the leech are produced oöcysts of a definite type. Furthermore, the schizogony takes place in

the red blood-corpuscles, which either circulate in the vessels or are held up in the capillaries of the internal organs. On this account the ordinary blood-films of the tortoise will show a variety of forms—schizonts in all stages of development from the merozoites onwards, and male and female gametocytes in various stages of growth. A similar cycle was proved for *H. nicoriæ* by Robertson, and the writer obtained evidence of a similar development in the case of the parasite of the tortoise, *Sternothærus adansonii*, of the Sudan (Plate XIX., 16-20, p. 1102). It is possibly safe to conjecture that the hæmogregarines of all aquatic tortoises, and possibly those of crocodiles, fish, and some amphibia, will be found to have a similar development in leeches. Laveran and Nègre (1905) examined ticks (*Hyalomma ægyptium*) taken off the land tortoise, *Testudo mauritanica*, which was infected with *Hæmogregarina mauritanica*. They discovered in the gut cysts containing sixteen sporozoites, which were similar to those which occur in the development of *H. stepanowi* in the leech, so that it is possible that the hæmogregarines of some of the land reptiles have a cycle of development in arthropods similar to that of *H. stepanowi* of the aquatic tortoise. On the other hand, the cysts may have been sporocysts which had escaped from oöcysts, in which case the development would correspond with that of *Hepatozoon muris*.

The small hæmogregarine of frogs, *Lankesterella minima* Chaussat, 1850, has been shown by Nöller (1913b) to develop only in the endothelial cells of the blood-vessels of the frog, and the sporozoites which are eventually produced in oöcysts within these cells enter the red blood-corpuscles and appear as small hæmogregarines (Fig. 380). These are transferred mechanically by leeches from one frog to another. On this account the parasite cannot be included in the genus *Hæmogregarina*. It is possible that other hæmogregarines of aquatic vertebrates, such as the small ones in newts, will be found to belong to this genus, but the majority of the forms which occur in the red blood-corpuscles of these animals resemble *H. stepanowi* rather than *L. minima*. They are frequently seen as large looped vermicules extending from one end of the red blood-corpuscle to the other, or as more compact forms which have been derived from the looped forms by fusion of the two limbs. In the latter nuclear multiplication takes place, and eventually merozoites are produced. Frequently the red cells containing the compact forms are held up in the internal organs, so that the schizogony stages are not seen in the peripheral circulation. In addition to these, the peripheral blood shows encapsuled sausage-shaped forms in various stages of growth. These are undoubtedly the developing gametocytes. To this group probably belong the large looped hæmogregarines of frogs and toads, the various forms in aquatic chelonians and crocodiles, and probably those of fish.

Hæmogregarines of Fish.

Hæmogregarines were first discovered in fish by Laveran and Mesnil (1901), who described *Hæmogregarina simondi* (Fig. 460) from the common sole (*Solea vulgaris*), and *H. bigemina* (Fig. 461) from the blenny (*Blennius pholis* and *B. montagui*). Since then numerous other forms have been described from marine fish, chiefly by Laveran and Mesnil, Brumpt and Lebailly, Neumann and Henry. Hæmogregarines occur much less frequently in fresh-water fish. *H. bettencourti* was described by França (1908*b*) from the eel (*Anguilla vulgaris*) of Portugal, and *H. nili* by the writer (1909) from *Ophriocephalus obscurus* of the Upper Nile, while Migone (1916*a*) observed certain forms in river fish of Paraguay. As they occur in the
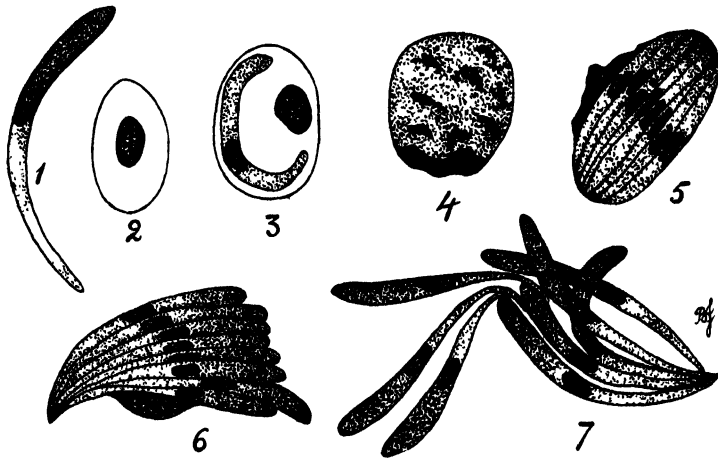
FIG. 460.—*Hæmogregarina simondi* FROM THE BLOOD OF THE SOLE, *Solea vulgaris* (× 1,800). (AFTER LAVERAN AND MESNIL, 1901.)

1-3. Vermicular forms free and in red blood-corpuscle and uninfected corpuscle.
4-7. Stages of schizogony in red blood-corpuscle.

blood the fish hæmogregarines resemble those of other animals, but little is known of their life-histories. Reproducing schizogony forms have been seen in the peripheral blood in some instances.

In the case of *H. bigemina* of *Blennius pholis* it appears that division into two takes place, as the hæmogregarine is often seen in pairs in the red cells, while other cells contain what appear to be dividing forms (Fig. 461). *H. quadrigemina* of *Callyonymus lyra* similarly divides into four, *H. simondi* of *Solea vulgaris* into eight (Fig. 460), and *H. polypartita* of *Gobius fraganellus* into sixteen. In these hæmogregarines it appears that the schizogony occurs in the red cells, which continue to circulate in the blood-vessels.

In the case of *H. æglefini* of the haddock (*Gadus æglefinus*), Henry noted in the blood certain structures in leucocytes of the mononuclear variety. These were in the form of spherical masses of cytoplasm, with one or more nuclei (Fig. 443). In the other cells occurred numerous small merozoite-like bodies. These structures were seen first by Neumann (1909), and were considered by him to represent a distinct parasite, which he named *Globidium multifidum*, a name subsequently changed to *Globidiellum multifidum* by Brumpt (1913c). They measure from 3 to 20 microns in diameter. Henry (1913a) considered the possibility of this organism being a leucocytozoon, but came to the conclusion that it was the schizogony stage of the hæmogregarine which occurred in the red blood-corpuscles. It seems possible that the cells are actually endothelial
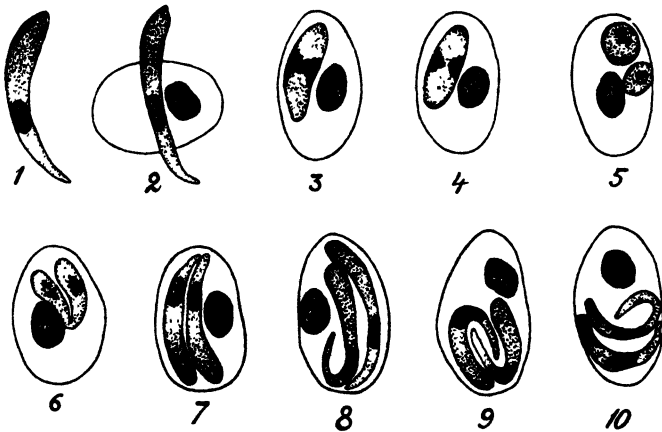
FIG. 461.—*Hæmogregarina bigemina* FROM THE BLOOD OF BLENNIES, *Blennius pholis* AND *B. montagui* (× 1,800). (AFTER LAVERAN AND MESNIL. 1901.)

1-2. Free forms in blood   3-5. Successive stages in division.
6-10. Growth of young forms into typical vermicules.

in origin. It has already been pointed out that many hæmogregarines undergo schizogony in these cells.

While studying *H. simondi* in the fresh blood, Henry noted that the hæmogregarines left the host cell, a process which occurs commonly amongst hæmogregarines observed *in vitro*, and moved about as vermicules. He described a process of granule-shedding wherein certain granules appeared to be extruded from the parasites. He compared these with the much-discussed " infective granules " of spirochætes, and concluded they were destined for further development. Within the red blood-corpuscles of *Cottus bubalis* and *C. scorpius* which harboured *H. cotti* were discovered small cytoplasmic bodies varying in length from 2 to 4·5 microns by 1 to 3 microns in breadth. These stained blue by

Romanowsky stains, and showed a marginal red-staining chromatic area. Many of them resembled the ring forms of species of *Plasmodium* or *Hæmoproteus*. Henry (1910) came to the conclusion that they were parasites distinct from the hæmogregarines, and proposed the name *Hæmohormidium cotti* (Fig. 443, 11-12). In his later contributions to the subject (1913a) he concluded that they represented the extruded granules of the hæmogregarines which had invaded the red cells in order to develop into the adult forms. There seems very little justification for such a conclusion, and it is highly improbable that the granules extruded from the hæmogregarines represent anything more than an excretory, if not a degenerative, process.

Another parasite of more definite structure was discovered in the mackerel (*Scomber scomber*) by Henry (1910). He proposed for it the name *Hæmatractidium scombri* (Fig. 443, 1-6). It occurs both in the red blood-corpuscles and leucocytes, and is mostly in the form of an elongated body, often closely applied to the nucleus. It is sometimes more pointed at one end than the other, and is possessed of a central nucleus. Figures illustrating degeneration of the red cells as a result of the presence of the parasite suggest that the so-called degenerate cells are actually damaged as a result of the process of spreading the film. The free forms, also, which may show pseudopodial prolongations, appear to be damaged specimens which have been dragged out of the cells that have been broken mechanically. The parasites look more like small hæmogregarines of the *Lankesterella* type than any other organism.

Franchini and Saini (1923) have described hæmogregarines from freshwater fish in France. A form in *Gobio fluviatilis* is named *Hæmogregarina gobionis;* one in *Cyprinus carpio, H. carpionis;* and one in *Tinca tinca, H. laverani.* The curious feature of these forms is that they were found chiefly in scrapings and sections of the wall of the intestine. In the blood the parasites were extremely rare, though in some cases there was intense infection of the gut, which showed not only free forms, but also numerous schizonts. It is far from clear that the forms seen in the blood were actually hæmogregarines, and the figures of these do not help in deciding this point. As regards the intestinal stages, these may equally well be developmental forms of coccidia. This seems all the more probable in view of the fact that the blood forms were so scanty, while the intestinal infection was a very heavy one. The remarks made regarding the sexual process in hæmogregarines and the presence of "infective granules," and the possibility of the latter pointing to a relationship between the trypanosomes and hæmogregarines of fish, show that the authors have no clear conception of these parasites.

Hæmogregarines of Land Reptiles.

A very large number of hæmogregarines has been described from land reptiles, lizards, snakes, and chelonians. The life-histories of these are not known except in the case of some of the parasites of lizards (*Lacerta*), as described by Reichenow, some of which belong to the genus *Schellackia*, and others to the genus *Karyolysus*. In the case of the former the red blood-corpuscles contain only sporozoites, while in the latter they contain gametocytes. Such a difference can only be detected when at least something of the life-history is known. At the present time, therefore, it is impossible to state whether a hæmogregarine of a reptile belongs to the first or second genus, or whether it belongs to either. The name *Karyolysus* was given to the hæmogregarine of species of *Lacerta* (*L. agilis, L. muralis, L. ocellata*) by Labbé (1894) because during the growth of the gametocytes within the nucleated red blood-corpuscles the nucleus tended to be fragmented. Whether this action on the nuclei is associated with a particular cycle of development cannot be stated with certainty. It hardly seems probable, so that one may expect that the majority of the numerous hæmogregarines of snakes and lizards will ultimately be found to belong to this genus. Some of them, however, will probably be included in the genus *Schellackia*, and for others new genera may have to be established. The discovery by Laveran and Nègre of cysts containing sixteen sporozoites in the tick *Hyalomma ægyptium* (? *H. syriacum*) suggests the possibility of some of the hæmogregarines of land reptiles belonging to the genus *Hæmogregarina* (see p. 1105).

In certain cases something is known of the asexual reproduction. That of *Karyolysus lacertarum* was first studied by Labbé, who found the encysted schizonts chiefly in the spleen. He noted two kinds of schizonts: one in which large merozoites (macromerozoites) are produced, and another in which small ones (micromerozoites) are formed. As has been pointed out above, the larger forms are probably the true merozoites, which again become schizonts, while the small ones are young gametocytes which enter the red blood-corpuscles (Fig. 457). Similar schizogony forms were seen by Laveran and Pettit (1909c) in the spleen and bone marrow of *Lacerta ocellata* infected with *Hæmogregarina curvirostris* Billet, 1904, while the writer (1909) described two distinct kinds of schizont in the liver of lizards (*Mabuia quinquetæniata*) infected with *H. gracilis* (Fig. 459). In the case of the last-named parasite the small merozoites were very narrow, and appeared in the red blood-corpuscles as narrow, elongate hæmogregarines (Plate XIX, 14-15, 1102, and Fig. 459). Shortt (1922a) described the schizogony of two hæmogregarines, which he discovered in two Persian lizards, as taking place in the lung. These

were *H. procteri* of *Phyllodactylus elisæ* (gecko) and *H. percomsi* of *Agama nupta* (rock lizard). Though in many cases there have been described two types of schizogony leading to the formation of macromerozoites and micromerozoites, these are by no means so sharply marked off from one another as the names imply. The two extremes are connected by many intermediate forms.

The schizogony stages of hæmogregarines of snakes have been described by various observers. They were first seen by Lutz (1901), while Laveran (1902c) described what were evidently the schizonts of *H. serpentium* in the lungs of the snake, *Eunectes murinus*. Here again there were two types of schizont producing large and small merozoites. The schizonts of the hæmogregarine of the spitting cobras of the Sudan, *Naja hajæ* and *N. nigricollis*, were discovered by the writer (1909) in the lungs, while Hartmann and Chagas (1910) observed schizonts of *H. serpentium* and *H. lutzi* in both the lungs and liver. Foley and Catanei (1925) have seen cysts containing from one to ten merozoites in the organs (lung, liver, spleen) of the viper (*Cerastes cornutus*) infected with *H. seurati* in Algiers. The cysts varied in size from 10 by 9 to 30 by 16 microns. The merozoites were all of one type.

As regards the transmitting hosts of the hæmogregarines of snakes little is known. It is probable that mites or ticks will be found to carry the infections. Prowazek (1908) examined specimens of *Porocephalus moniliformis* from the lungs of a python which harboured hæmogregarines. In the gut of the *Porocephalus* he found vermicular forms and cysts containing a mass of cytoplasm with a single nucleus. He considered these to be developmental stages of the hæmogregarine, and the *Porocephalus* to be its transmitting host. Patton (1908) discovered cysts in *P. pattoni*, a linguatulid found in the lungs of *Zamenis mucosus*. Each cyst included a number of smaller cysts, within which were spindle-like structures (sporozoites ?). Patton concluded they were encysted stages of a parasite peculiar to the *Porocephalus*, which had nothing to do with the hæmogregarine. No evidence has yet been produced that the *Porocephalus* plays the part of a true transmitting host, whatever may be the nature of the cysts described.

Hæmogregarines of Mammals and Birds.

Hæmogregarines of mammals were first made known in 1905, when Balfour discovered *Hepatozoon balfouri* of *Jaculus gordoni* of the Sudan. In the same year Laveran saw the parasite (named by him *Hæmogregarina balfouri*) in the *J. orientalis* of Tunis. These hæmogregarines, which are included in the genus *Hepatozoon*, are parasitic in the red blood-corpuscles of the jerboas. A number of other similar forms has been described.

Hæmogregarines of the Red Blood-Corpuscles of Mammals.

Hepatozoon balfouri Laveran, 1905 (*H. jaculi* Balfour, 1905): *Jaculus orientalis* (jerboa), Tunis; *Jaculus jaculus* (jerboa), Sudan.

H. sp. Rodhain, Pons, Vandenbranden and Bequaert, 1913: *Jaculus jaculus* (jerboa), Belgian Congo.

H. gerbilli Christophers, 1905: *Gerbillus indicus* (gerbil), India.

H. dasyuri Welsh, Dalyell and Burfitt, 1909: *Dasyurus viverrinus* (dasyure), Australia.

H. petauri Welsh and Barling, 1909: *Petaurus sciureus* (flying opossum), Australia.

H. peramelis Welsh and Dalyell, 1910: *Perameles nasuta* (bandicoot), Australia.

H. sp. Kleine, 1910: *Dendromys insignis* (mouse), Tanganyika.

H. sp. Rodhain, Pons, Vandenbranden and Bequaert, 1913: *Petrodromus tetra-dactylus* (elephant shrew), Belgian Congo.

H. sp. Rodhain, 1915: *Cricetomys gambianus* (giant rat), Belgian Congo.

H. didelphydis d'Utra, Silva, and Arantes, 1916: *Didelphys didelphys aurita* (opossum), Brazil.

In addition to the various species of *Hepatozoon* occurring in the red blood-corpuscles, others inhabit the leucocytes. *H. muris* was the first of these to be described by Balfour (1905) from *Rattus norvegicus* of the Sudan. In the same year *H. canis* was described from the dog by Bentley and James in India. These two forms have a wide distribution, and have been observed in many parts of the world. Other species are also known.

Hæmogregarines of the Leucocytes of Mammals.

Hepatozoon muris Balfour, 1905: *Rattus norvegicus* (brown rat), Sudan.

  = *H. ratti* Adie, 1907: *Rattus rattus* (black rat), India.

  = *H. innoxium* Kusama, Kasai and Kobayashi, 1919: *Rattus rattus alexandrinus* (rat), Japan.

  = *H. perniciosum* Miller, 1908: *Rattus rattus* (white rat), America.

H. canis Bentley and James, 1905: *Canis familiaris* (dog), India.

H. funambuli Patton, 1906: *Funambulus pennantii* (palm squirrel), India.

H. felis Patton, 1908: *Felis domesticus* (cat), India.

H. leporis Patton, 1908: *Lepus nigricollis* (hare), India.

H. musculi Porter, 1908: *Mus musculus* (white mouse), England.

H. rotundatum Patton, 1910: *Canis aureus* (jackal), India.

H. canis adusti, Nuttall, 1910: *Canis adustus* (jackal), East Africa.

H. citellicolum Wellman and Wherry, 1910: *Otospermophilus beecheyi* (ground squirrel), California.

H. criceti Nöller, 1912: *Cricetus frumentarius* (hamster), Austria.

H. sylvatici Coles, 1914: *Mus (Apodemus) sylvaticus* (wood mouse), England.

H. sp. Kleine, 1910: *Mus (Pseudomys) cunninghami* (mouse), Tanganyika.

H. chattoni Leger, 1912: *Hyæna crocuta* (hyena), Senegal.

H. rotundatum var. *canis familiaris* Martoglio, 1913: *Canis familiaris* (dog), Eritrea.

H. plicatum marmotæ Martoglio, 1913: *Arctomys marmota* (marmot), Eritrea.

H. arvalis Martoglio, 1913: *Arvicola (Microtus) arvalis* (field vole), Somaliland.

H. microti Coles, 1914: *Microtus agrestis* (field vole), England.

H. pitymysi Splendore, 1918: *Pitymys savi* (field vole), Italy.

H. cuniculi Sangiorgi, 1914: *Oryctologus (Lepus) cuniculus* (rabbit), Italy.

H. akodoni Carini and Maciel, 1915: *Akodon fuliginosus* (vole-mouse), Brazil.
H. gœtulum Sergent, 1921: *Atlantoxerus gœtulus* (ground squirrel), North Africa.
H. sp. Fantham, 1920: *Giraffa* (giraffe), South Africa.
H. sp. Fantham, 1921: *Cervicapra arundinum* (reed buck), South Africa.

The development of these hæmogregarines, whether they inhabit red cells or leucocytes, is very uniform. Schizogony takes place in the internal organs (liver, lung, spleen, and bone marrow), and usually in endothelial cells of the blood-vessels. In some cases, as in *H. balfouri* of the jerboa, the schizonts appear to be in the glandular cells of the liver. As a rule, two main types of schizogony can be distinguished. In one a small number of large merozoites is produced, while in the other there is a large number of small ones. The former appear to be the true asexual forms, which by infection of other cells of the same type lead to further multiplication by schizogony. The small forms represent young gametocytes, which enter the red blood-corpuscles or leucocytes. Schizonts producing a varying number of merozoites of intermediate size also occur. The schizonts do not appear in the peripheral blood, so that the forms found there are only gametocytes destined for development in the invertebrate.

The invertebrate cycles of the mammalian hæmogregarines, wherever they are known, agree very closely with those of *H. muris* and *H. canis* described above (Figs. 453, 455.) After fertilization, the details of which are not accurately known, the zygote increases enormously in size in the tissues of the invertebrate, and the oöcyst produces within it a number of sporocysts, in each of which sporozoites are developed. It is probable that infection of the vertebrate takes place through the invertebrate being eaten by the vertebrate. Sporogony cycles of this kind have been studied by Miller (1908) and Kusama, Kasai and Kobayashi (1919) in the case of *H. muris* in the mite, *Lælaps echidninus;* by Christophers (1906, 1907a, 1912) and the writer (1911a) for *H. canis* in the tick, *Rhipicephalus sanguineus* (Fig. 455); and by Christophers (1905) for *H. gerbilli* in the louse, *Hæmatopinus stephensi.* Nöller (1912c) studied *H. criceti* of the hamster (*Cricetus frumentarius*). It was seen only within leucocytes obtained from an abscess of the ear. Mites (*Lælaps echidninus*) showed cysts (probably sporocysts) containing sixteen sporozoites, and these Nöller considered to be derived from the hæmogregarines. Splendore (1918, 1920) found that *H. pitymysi* could complete its development in three ectoparasites of the field vole (*Pitymys savii*). These are two fleas (*Ceratophyllus fasciatus* and *Typhlopsylla asimilis*) and a louse (*Hoplopleura acanthopus*). Field voles were readily infected by causing them to eat the tissues of the infected arthropods. An attempt was made to infect these animals with *H. muris* by feeding them with infected

mites (*Lœlaps echidninus*) taken from rats (*Rattus norvegicus*). No infection occurred, a fact which lends support to the view that *H. pitymysi* and *H. muris* are distinct parasites. In the case of *H. balfouri* of the jerboa, Balfour (1906) mentioned the occurrence of spherical cysts 16·4 to 25·6 microns in diameter in *Pulex cleopatræ* taken from infected animals. It is possible they were sporocysts of *H. balfouri*, but no details of their structure were given. An examination of mites (*Dermanyssus gallinæ*) from the animals did not reveal any developmental stages. As regards the hæmogregarines of birds, these have been fully discussed above (pp. 1042 and 1095).

Supposed Hæmogregarines of Man, Monkeys, and Ox.

In this place may be considered certain organisms which have been erroneously described as hæmogregarines from man, monkeys, and the ox.

The first of these to be regarded as a hæmogregarine of man was found by Krempf (1917) in smears of spleen puncture material obtained from a young China-man suffering from fever associated with splenomegaly. The organism was present in very small numbers, and is described as occurring either in red blood-corpuscles or free (Fig. 462, 1-4). It was in the form of an elongate blue-staining structure with a central red-staining granular nucleus. The cytoplasm was vacuolated in some of the specimens. The length varied from 6 to 20 microns and the breadth was 1·5 microns, the longer individuals being doubled in the form of a loop, as occurs commonly in hæmogregarines. No developmental stages were found, nor was the organism seen in the peripheral blood. The longest forms contained two nuclei, and were said to divide by transverse fission. Krempf gave it the name *Hæmogregarina hominis*.

The second human hæmogregarine was recorded by Roubaud (1919) from the blood of a woman who was about to return to the Congo after a stay of four years in Paris (Fig. 462, 5-15). A blood-film was made, and it was in this single film that the organism described was found. Further examinations of the blood on the following day revealed nothing abnormal. As in Krempf's case, the organism is said to be either within red blood-corpuscles or free. Looped individuals were also described. The large forms measured from 9 to 11 microns in length, with a breadth of 2·8 to 3·5 microns. Roubaud describes these forms as having the classical structure, but it must be admitted that the nuclei do not resemble the nuclei of typical hæmogregarines as they occur in blood-films. Roubaud named the organism *H. inexpectata* (Fig. 462, 5-15). Lebœuf (1921) claims to have seen the same organisms in spleen smears made from a girl suffering from splenomegaly in the Belgian Congo.

The third form was discovered by Sergent, Ed. and Et., and Parrot (1922) in the blood of a girl in Corsica (Fig. 462, 16-24). It was seen in one thin film, and in three thick films made from finger blood during the course of a malarial survey. In the thin film sixteen parasites were found, and of these only two were within the red cells. The authors remark that they resemble atypical hæmogregarines, and propose to name the organism *H. elliptica*.

Noc (1922) records as *H. gallica* certain structures which he found in small numbers in blood-films of a man fifty-nine years of age who had lived all his life in the neighbourhood of Paris. The patient suffered from a profound anæmia of pernicious type. A microphotograph of the supposed hæmogregarine shows
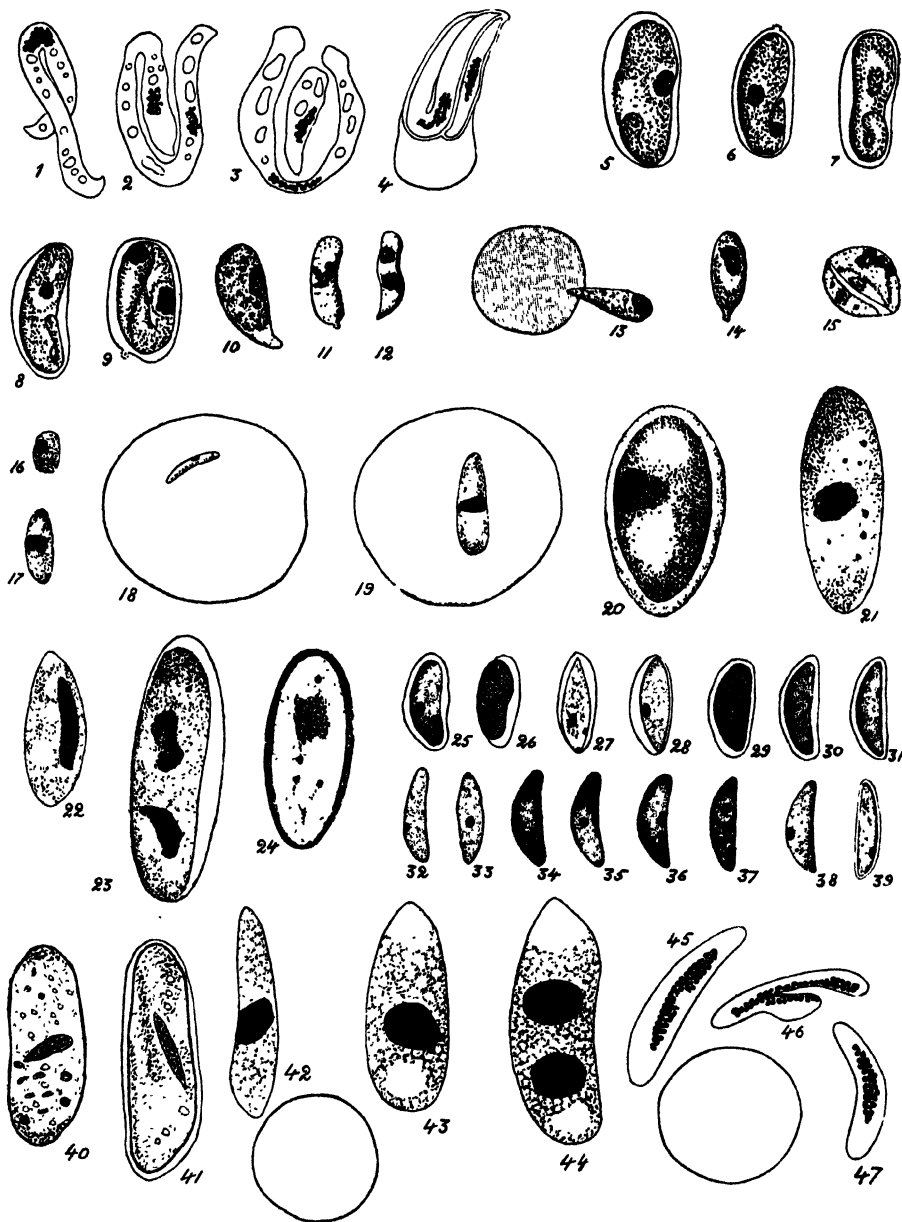
FIG. 462.—SUPPOSED HÆMOGREGARINES OF MAN, MONKEY, AND OX. (FROM THE *Tropical Diseases Bulletin*, VOL. XX., 1923, AFTER THE RESPECTIVE AUTHORS.)

1-4. Krempf's *Hæmogregarina hominis*.   5-15. Roubaud's *Hæmogregarina inexpectata*.
16-24. Sergent's *Hæmogregarina elliptica*.
25-39. Nattan-Larrier's *Hæmogregarina equatorialis*.
40-41. Langeron's *Hæmogregarina cynomolgi*.   42-44. Legras's *Hæmogregarina boum*.
44-47. Martoglio and Carpano's *Hæmogregarina bovis*.

a sausage-shaped body amongst a group of red cells. It measured 19·8 microns in length by 4·5 in breadth. It was bluntly pointed at one end, while the other end is described as terminating in a turned-up loop. It contained two bodies described as nuclei. At the second examination of the blood two vermicular structures measuring 15 by 5 microns were seen. Each contained a chromatic granule. In addition were found what are called division forms in the shape of curved merozoites measuring 5 to 6 by 1·5 microns. Two such bodies, or possibly three, were together, accompanied by a spherical mass 1·5 microns in diameter described as a residual body. The patient died, and it was not possible to have an autopsy. After discussing the various " hæmogregarines " which have been noted in man, Noc concludes that this one is a new species. Roubaud, however, remarks during a discussion following the presentation of Noc's paper that he believes it to be the same as the form (*H. inexpectata*) seen by him.

Nattan-Larrier (1922, 1923) describes as *Hæmogregarina equatorialis* certain curved bodies which he found in 1906 in the blood of a patient who had recently returned from the Congo suffering from trypanosomiasis (Fig. 462, 25-39). The bodies were found on only one occasion, and were fairly numerous. They were extracellular, resembled a lentil in shape, had rounded ends, and measured on an average from 6 to 7·5 by 1·6 to 2·4 microns. The nucleus was central in position, had at most a diameter of half that of the parasite, and was placed against its convex side. In addition some very much smaller bodies, which were regarded as possible developmental stages of the larger ones, also occurred in the films. Nattan-Larrier regards these bodies as similar to *H. elliptica* of Sergent and Parrot, but sufficiently different to justify the new name proposed by him.

Blanchard and Langeron (1913),during an investigation of the malarial parasite, *Plasmodium cynomolgi* of *Macacus cynomolgus*, described certain structures which they had found in the blood of an animal which had died of the plasmodial infection (Fig. 462, 40-41). The description of hæmogregarines in man by Krempf and Roubaud convinced Langeron (1920) that the structures originally described were also hæmogregarines. He proposed the name *Hæmogregarina blanchardi*, but as this was preoccupied he (1920a) substituted the name *H. cynomolgi*. It has a length of 17 to 19 microns and a breadth of 5 to 6 microns. The cytoplasm stains intensely blue, and there is a central red-staining nuclear area. Some of the forms appear to be free in the plasma. Others are included in what are interpreted to be red blood-corpuscles. Leger, M., and Bédier (1922) state that they have found a very similar form in a baboon (*Papio sphinx*) in the French Sudan. They name the organism, which they consider a hæmogregarine, *Hæmogregarina cynomolgi* var. *papio*. It is remarkable that both the baboon and the monkey which Langeron examined were infected with *Plasmodium kochi*, so that the possibility of the supposed hæmogregarine being an abnormal form of this parasite has to be considered.

The writer (1923) has examined the claims made by the above-mentioned observers, and has come to the conclusion that all these supposed hæmogregarines are actually vegetable cells of extraneous origin. Though the various structures possibly bear some slight resemblance to hæmogregarines, their inclusion in this group is at present quite unjustifiable. No known hæmogregarine has such long vermicular stages as Krempf depicts, and in none of the supposed hæmogregarines is the nuclear structure like that of true hæmogregarines as seen in blood-films. Krempf's statement that certain forms contain two nuclei and divide by transverse division is direct evidence against their hæmogregarine nature. Roubaud's parasite was seen in only one film, and subsequent blood examinations did not reveal any. This is quite contrary to the behaviour of hæmogregarines, which at any rate persist in the blood for some time. The possibility of an extraneous origin has to be con-

sidered, and some of the bodies resemble in many respects large yeasts or vegetable organisms like *Polytoma uvella*, which may contaminate blood-films or the liquids used in the staining process, and which, when stained, show a blue cytoplasm and bright red nucleus which is quite unlike that of hæmogregarines. The fact that the enclosing membrane, supposed to be a red cell, possessed a knob in some cases (Fig. 462, 10-11) rather suggests the sheath of a vegetable organism, for such a structure occurs on the membrane of *P. uvella*. Balfour (1911), in a paper on fallacies and puzzles associated with blood work, gives a figure of a blood-film made from a cob which had undoubtedly been contaminated with intestinal organisms, some of which bear an even more striking resemblance to hæmogregarines than the structures which have just been described (Fig. 433, E. 3). As to the nature of Krempf's parasite it is difficult to form any opinion, though to conclude from the evidence available that it is a hæmogregarine is certainly unjustifiable. It is very probably a coiled filament of vegetable nature. Similarly, the form described by the Sergents and Parrot seems too atypical to be regarded as a hæmogregarine without further evidence. The majority of the bodies are admittedly extracellular, while the presence of the thickened capsule is quite unlike anything occurring in hæmogregarines. As the capsule was present in the dehæmoglobinized thick films it could not be the remains of the red cell.

The structures described by Nattan-Larrier as hæmogregarines are certainly not organisms of this nature. They are most probably yeasts. Similarly, Noc's supposed hæmogregarine is probably not a Protozoon at all, but a vegetable cell. Langeron's parasite of the monkey is still more doubtful, and it must be remembered it occurred in an animal which had died of another infection. Like the form described by Leger and Bédier from the baboon, it probably represents a vegetable organism. The writer is convinced, therefore, that hæmogregarines of human beings and monkeys have still to be discovered. It would be expected that if such parasites did occur, they would resemble the leucocytic forms so common in mammals (dog, rat, etc.), or possibly the rod blood-corpuscle type seen in the jerboa.

Mention must also be made of two supposed hæmogregarines of cattle. The first of these was described by Martoglio and Carpano (1906), and was named *Hæmogregarina bovis* (Fig. 462, 45-47). It occurred as elongate, slightly curved bodies, measuring 7 to 10 by 1·6 to 2 microns, which were found in wet blood-films made from an ox. The films were smeared and stained next day, and the bodies appeared to have blue cytoplasm and elongate granular red nuclei. Further films made from the animal failed to reveal the parasites. There is little doubt that the bodies were contaminating vegetable organisms. Legras (1918) described as *Hæmogregarina boum* certain ovoid bodies measuring 6·8 to 8·6 by 1·5 to 2·5 microns which occurred in blood-films made from a heifer in Algeria (Fig. 462, 42-44). The cytoplasm was blue, and each body contained one or two red-staining areas and terminal vacuoles. As in the case of the bodies described by Martoglio and Carpano, they were extracellular. There is no evidence whatever that they were Protozoa, much less hæmogregarines, and it is safe to assume that they were vegetable cells which had found their way into the films.

The writer (1923) pointed out that the large vegetable organisms which occur in the fæces of rabbits, when stained in blood-films, bear a resemblance to hæmogregarines (Plate III., 3, p. 394). If some of the fæces containing these organisms be mixed with water and allowed to sediment for a few minutes, the vegetable cells will remain in the supernatant fluid. A thin film of this fluid is spread on a clean slide and allowed to dry. Over this a blood-film is made in the usual manner. After staining with Leishman stain, it will be found that the sausage-shaped vegetable cells have blue cytoplasm and red nuclei, and afford a demonstration of the manner

PLATE XX.
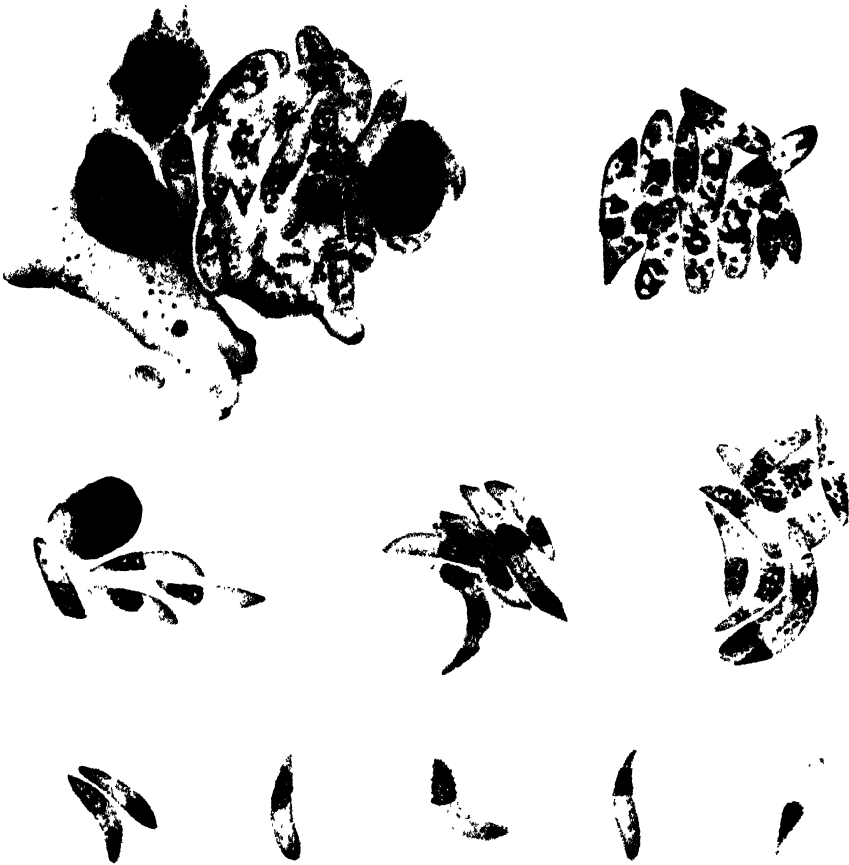
Merozoites of a Sporozoan parasite discovered in smears of spleen puncture material from a case of splenomegaly in the Sudan (x,1500). (After Archibald and Susu, 1924.)

in which such cells may simulate hæmogregarines and lead to errors in diagnosis when blood-films are contaminated. Similarly, other vegetable organisms, when they contaminate blood-films, may resemble developmental stages of Sporozoa (Plate III., 4-6, p. 394, and Fig. 433).

Sinton (1925) has found in films of human blood bodies which resemble the " hæmogregarines " of Roubaud, the Sergents and Parrot, Langeron and Legras. He gives good coloured drawings of the organisms, but recognizes their vegetable nature, and points out that they probably got into the films from an old supply of distilled water used in staining. He agrees with the writer that the so-called hæmogregarines of man are vegetable organisms.

Sporozoon of Undetermined Genus in Human Spleen.

Archibald and Susu (1924) discovered what appear to be the schizogony stages of a sporozoon in spleen smears of a case of splenomegaly in the Sudan. The smears, which the writer had the privilege of examining, contained isolated bodies like merozoites, and several clusters of these, which are best interpreted as groups of merozoites resulting from schizogony (Plate XX., p. 1116). No other stages were found in the films, which were made by spleen puncture. It is possible the organism was a coccidium, and that the forms in the spleen represented parasites which had found their way to this organ from the intestine, which, however, was not examined. The possibility of the parasite being a hæmogregarine has also to be considered, though hæmogregarines were not found in the peripheral blood, which was carefully examined. It is impossible, however, from the data available to place the parasite in any genus.

2. SUB-CLASS: Gregarinina.

The members of this sub-class are commonly known as gregarines, and are found in invertebrate animals and ascidians. They were first seen and described by Léon Dufour, who gave them the name *Gregarina* in 1828. Von Siebold (1839) gave the name pseudonavicellæ to the spores which occurred in cysts, and which had previously been seen by Henle (1835) in the vesiculæ seminalis of earth-worms. Kölliker (1845 and 1848) first recognized that gregarines were unicellular animals, while Stein (1848) demonstrated that the pseudonavicellæ were derived from them. Though various observers studied these parasites, Wolters (1891) was the first to give any detailed account of their development. He noted that two gregarines became encysted together, but erroneously concluded that after the nucleus of each had divided off a reduction body, fusion of the gregarines and their nuclei took place. The resulting zygote was then supposed to divide up into sporoblasts. Wolters's account was generally accepted till Siedlecki (1899) for *Lankesteria ascidiæ*, Cuénot (1901) for *Monocystis agilis*, and Léger, L. (1902), for *Stylorhynchus longicollis* gave

correct accounts of the development. These observers showed that the two gregarines in each cyst did not unite, as Wolters had stated, but that after repeated nuclear divisions each gave rise to a number of gametes which united in pairs to form zygotes. Each zygote then became encysted to form the characteristic spore.

A typical gregarine commences its growing or trophic phase as an intracellular parasite chiefly of the intestinal epithelium (Fig. 463). After a certain period of growth it protrudes from the host cell, and eventually leaves it for the lumen of the gut or body cavity, in which it leads a free-living existence, moving about more or less actively amongst the contents. In this condition it is usually an elongate body, which may attain what is an enormous size for a Protozoon. Small forms only 10 microns in length occur, but others may reach a length of 16 mm. (16,000 microns), or over ½ inch. The body consists of a cuticle, ectoplasm, and endoplasm. The cuticle often shows striations running in a longitudinal direction. The ectoplasm is clear and hyaline, but in those forms endowed with powers of active movement the deeper part contains contractile fibres or myonemes which form a distinct layer, the *myocyte*, the outer clear part being known as the *sarcocyte* (Fig. 28, A). The more liquid endoplasm is generally filled with numbers

FIG. 463.—SECTION OF INTESTINE OF MEAL-WORM (*Tenebrio molitor*), SHOWING GREGARINES IN AND ATTACHED TO EPITHELIUM AND FREE IN LUMEN (×300). (AFTER L. PFEIFFER, 1893.)

of granules of varying size, giving the gregarine a very opaque appearance. The largest of these may be several microns in diameter, and they consist of paraglycogen, a substance allied to glycogen. Within the endoplasm lies the large vesicular nucleus, composed of a spherical membrane enclosing nuclear sap, a loose network, and one or more large karyosomes.

In a certain small group of gregarines (SCHIZOGREGARINIDA) the free-living adult gregarine multiplies by a process of schizogony, producing merozoites, which enter other epithelial cells and again grow into adults.

The great majority of gregarines (EUGREGARINIDA) do not multiply asexually by schizogony, but as soon as they are full grown they commence a sexual reproduction, which terminates in the formation of

oöcysts and sporozoites. In those cases, therefore, in which schizogony does not occur, every gregarine present becomes a gametocyte and is the result of growth from a sporozoite. The oöcysts produced escape from the body of the host, and are taken up casually by another host with its food. The sporozoites escape and enter epithelial cells, where they become gregarines, as described above, and enter once more on the sexual phase. with the production of oöcysts. In consequence of this loss of asexual reproduction there can be no multiplication of the gregarines in any host, and as a result the hosts do not become so intensely infected as is the case with coccidia, which reproduce asexually for several generations before the sexual phase occurs. In the sexual reproduction of the gregarines two free forms, either in the gut or body cavity, come together and associate without fusion of their cytoplasm. The two become surrounded by a cyst, which may be termed the *gametocyst* (Fig. 464). Within this cyst each gregarine, which is actually a gametocyte. divides into a number of gametes (often called primary sporoblasts).

FIG. 464.--LARVA OF *Tipula oleracea* WITH OPENED BODY, SHOWING NUMEROUS GAMETOCYSTS OF A GREGARINE ON THE EXTERNAL SURFACE OF THE INTESTINE (× *ca*. 5). (AFTER LÉGER, 1892.)

The gametes derived from one gregarine then unite with those from the other, and give rise to a number of zygotes (secondary sporoblasts). These zygotes then become enclosed in secondary cysts, which are usually called sporocysts, but which on the terminology adopted for the coccidia should be termed oöcysts. Within these secondary cysts, or oöcysts, the zygote divides to produce as a rule eight sporozoites. From the two gregarines which originally associated there is thus produced a gametocyst containing numbers of smaller oöcysts, in each of which occur eight sporozoites. The still intact gametocyts may be passed out of the intestine, or it may rupture in the gut and liberate the oöcysts. In any case, it is the oöcyst containing the eight sporozoites which carries the infection from one host to another. Theoretically, an animal ingesting one of these secondary cysts has liberated into its gut eight sporozoites. These enter epithelial cells and become eight gregarines, which can produce four couples, and thus four gametocysts, within each of which are formed many oöcysts. The oöcysts are passed out of the body, and unless the
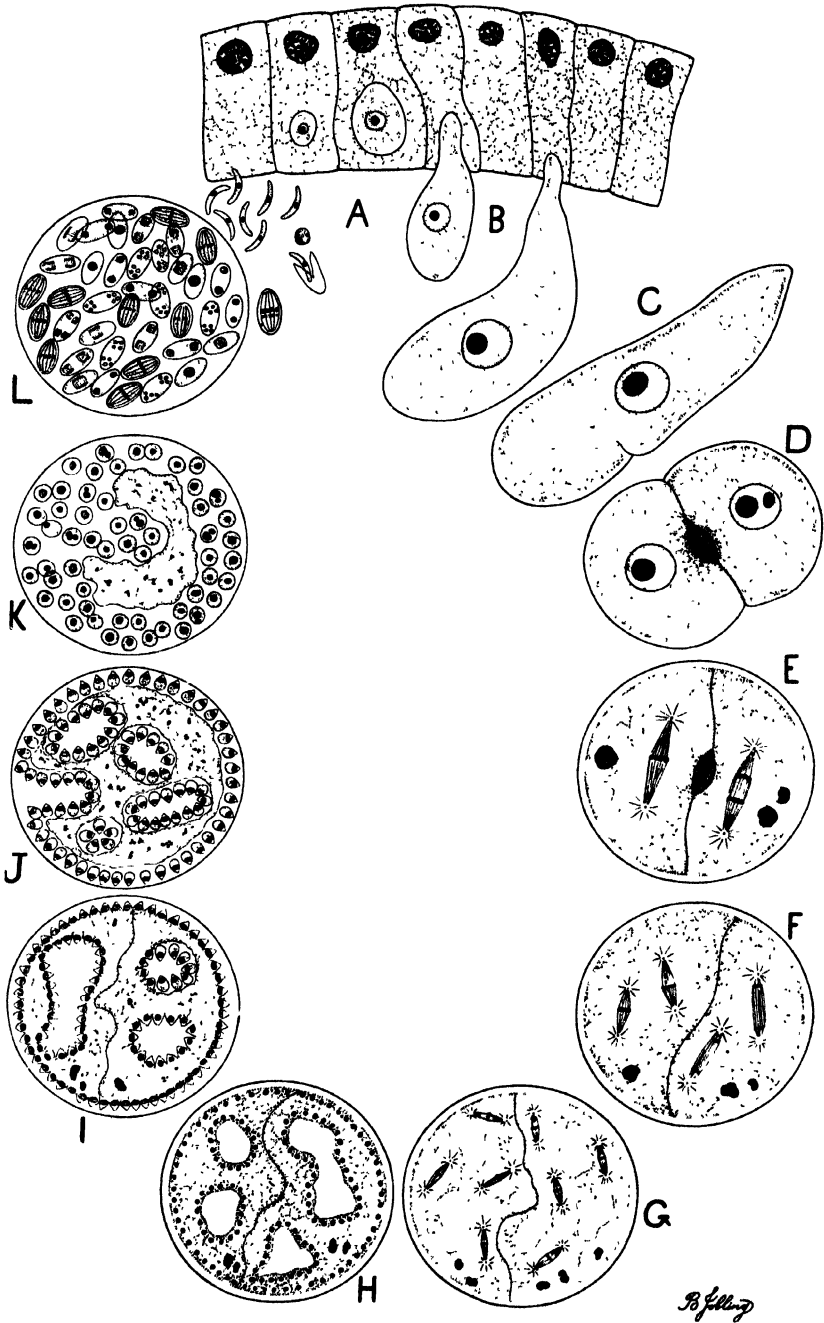
FIG. 465.—DIAGRAM OF LIFE-CYCLE OF GREGARINE OF THE TYPE *Lankesteria culicis*
(× *ca.* 1,000).   (ORIGINAL.)

[*For description see opposite page.*

animal ingests other oöcysts its infection is automatically brought to an end. On the other hand, an animal eating a single oöcyst of an *Eimeria* also has liberated into its gut eight sporozoites, but these commence to reproduce asexually by schizogony, leading to an enormous increase of parasites before the sexual phase is initiated.

Lankesteria culicis (Ross, 1898).—As an illustration of the life-history of a gregarine the parasite *L. culicis*, which is often found in the yellow fever mosquito, *Aëdes argenteus* (*Stegomyia fasciata*), may conveniently be considered (Figs. 465 and 561). The parasite was first seen by Ross (1895) in India, and referred to as *Gregarina culicidis* in this year, and later (1898a) as *L. culicis*. It was rediscovered by Marchoux, Salimbeni and Simond (1903) in South America, and by Bacot (1916) and Macfie (1917a) in West Africa. The writer (1911a) studied the gregarine in Bagdad, and found that it belonged to the genus *Lankesteria*.

The infection is commenced by the larva eating the oöcysts which have been deposited in the water by adult mosquitoes (Fig. 465, A). In the gut of the larva the eight sporozoites which escape enter the epithelial cells of the stomach and grow into gregarines, which eventually become free and move about amongst the intestinal contents (Fig. 465, A-C). When the larva becomes a pupa it ceases to take food, and the gregarines leave the gut and enter the Malpighian tubes, within which they associate in pairs and produce large gametocysts (Fig. 465, D). Within these cysts each gregarine divides into gametes, which conjugate in pairs to produce zygotes. These in their turn become encysted in oöcysts, within which each segments into eight sporozoites (Fig. 465, E-L.) While this development has been proceeding, the adult mosquito hatches from the pupal case. The Malpighian tubes of an infected adult mosquito will be found to contain gametocysts filled with oöcysts. The gametocysts then rupture, the oöcysts escape into the cavity of the Malpighian tubes, and make their way to the intestine, whence they pass into water to await ingestion by other larvæ.

The gregarine has been recorded from various parts of the world, and will probably be found to occur wherever the particular mosquito is found.

A. Escape of eight sporozoites from oöcyst and infection of the intestinal cells of mosquito larva.
B. Growth of young gregarine till it protrudes from the cell though remaining attached.
C. Gregarine which has become free in lumen of intestine, whence it migrates to the Malpighian tubes.   D. Association of two gregarines in Malpighian tubes.
E-G. Associated gregarines in gametocyst showing nuclear multiplication.
H. Nuclear multiplication completed and gamete nuclei arranged on surface of body and vacuoles which have formed.
I. Commencing formation of gametes by budding process.
J. Gametes have been formed and a large residual body has been left over.
K. Conjugation of gametes, which are of two kinds as regards the size of their nuclei.
L. The zygotes have become elongated and encysted in oöcysts in which nuclear multiplication to form eight nuclei and division of the cytoplasm into eight sporozoites is taking place.

In any locality there are special breeding places which are infected. The writer noted that a high percentage of the larvæ from a particular well in Bagdad was infected, while in another similar well the parasites were not to be found. This is probably dependent upon the fact that the mosquitoes do not travel far, and return to the water from which they were hatched to lay their eggs and die.

As already explained, the infection is initiated by the larval mosquito ingesting an oöcyst which has been deposited in the water by an adult mosquito. Within the gut the oöcyst ruptures, and the eight sporozoites escape and penetrate the epithelial cells of the intestine, where they become more or less spherical intracellular parasites. This process has not actually been observed in the case of *L. culicis*, but has been seen to happen in the case of other similar gregarines. Within the epithelium the gregarine increases in size, and at the same time expands and destroys the cell in which it lives. It becomes an elongate body with a large central nucleus. It lies in a vacuole in the enlarged cell, and at one end has an organ (*epimerite*) by means of which it remains attached at one point to the cytoplasm of the cell. From this organ of attachment, which is much more highly developed and complicated in some other gregarines, there radiate through the body of the gregarine certain fibres which are possibly contractile and of the nature of myonemes. Eventually the gregarine becomes so large that the cell ruptures, and it hangs into the gut lumen still attached, however, to the degenerate remains of its host cell. These fully-grown forms are usually about 50 microns in length, though Ross, who first discovered this gregarine in India, mentions 200 microns as the maximum. The full-grown gregarine remains attached to the gut wall for some time, and then falls into the gut lumen, where it moves about amongst the contents. These movements, which result from the contractions of the myonemes in the inner layer of the ectoplasm, are of three kinds. The gregarine may bend and then straighten itself; it may produce constrictions, which pass in peristaltic waves from one end of its body to the other, or it may progress through the medium in which it lives without any apparent movement of contraction, much as a slug glides over the surface of any object. These free-living forms, about 50 microns in length, have a granular cytoplasm, while at the anterior end the remains of the organ of fixation or epimerite can be detected. At the centre of the body is the large spherical nucleus. It has a nuclear membrane, within which is a space filled with fluid traversed by a linin network. Usually there is a single large karyosome, which in stained specimens may be seen to possess one or more vacuoles. Sometimes several karyosomes are present. Nearly all the chromatin material of the nucleus is contained within the karyosome, but there are some fine

granules distributed upon the nuclear membrane and upon the linin network.

The next stage in the life-history of *L. culicis* is initiated by the pupation of the larva. When this occurs, the gregarines actively migrate from the gut to the Malpighian tubes. They pass into these and associate in pairs, each pair surrounding itself with a gametocyst, which is a delicate transparent membrane. There may be as many as a dozen such cysts in a single Malpighian tube, which is dilated by their presence, while the cells are distorted. The gregarines, when they unite in pairs, become attached to one another by their extreme anterior ends, the remains of the organs of fixation which previously served to attach them to the gut epithelium now serving to attach them to one another. The two gregarines then by contraction form a mass which is more or less spherical, so that the gametocyst is also spherical. In sections of a Malpighian tube at this stage the spherical cysts can be seen enclosing two gregarines. The original point of attachment of the gregarines stains deeply, and often resembles a nucleus. In each gregarine there is at first a single nucleus, which commences to change in character soon after formation of the gametocyst. This process has been more fully studied in the case of other gregarines, but there are indications that similar changes take place in the case of *L. culicis*. The nuclear membrane gradually disappears, and the karyosome, which has become vacuolated, fragments. Two centrosomes appear in the neighbourhood of the nucleus, and there is formed between them a spindle—the first nuclear spindle—upon the fibres of which becomes arranged in the form of chromosomes some of the chromatin material of the nucleus. The bulk of the chromatin contained in the karyosome appears to be unused, and ultimately degenerates and disappears with the karyosome. As a result of this division there are formed two nuclei, each of which proceeds to divide again by a similar process of mitosis. In this way, by repeated mitotic divisions, the number of nuclei in each gregarine is increased. The successive divisions of the nuclei in each gregarine take place simultaneously, so that the number of nuclei in the two gregarines at any moment is the same. Finally, nuclear multiplication comes to an end, when each gregarine may contain some hundreds of nuclei. The details of the nuclear divisions and the behaviour of the chromosomes have not been followed in *L. culicis*, but in other gregarines, as explained above, very interesting changes have been found to take place (pp. 91 and 108).

When nuclear multiplication has ceased the nuclei pass to the surface of the gregarine, and there are formed small protoplasmic elevations or buds, into each of which passes one of the nuclei. The small elevations are then separated from the gregarines and become the gametes. When the

number of the nuclei is large, there may not be sufficient surface accommodation for all. Accordingly, it frequently happens that a number of large vacuoles appear in the cytoplasm, and upon the surface of these vacuoles many of the nuclei take up a position, with the result that gametes are budded off into the vacuoles. Gamete formation occurs in the two gregarines at the same time. When it is complete there is left over from each gregarine a mass of cytoplasm, the residual body, which contains a number of unused nuclei and the remains of the karyosome of the original gregarine nucleus. The residual mass gradually disintegrates. In the case of *L. culicis* the gametes produced by the two gregarines are of the same size, but they differ as regards their nuclei. The gametes produced by one of the gregarines have large nuclei and are to be distinguished as female gametes, the gregarine producing them being a female gametocyte. The gametes with smaller nuclei are male gametes, and are produced by the other gregarine, which is a male gametocyte. Within the gametocyst the gametes conjugate in pairs, and during the process each gamete develops a conical elevation on the side of the body near which the nucleus lies. The gametes apparently become attached to one another by this elevation, and when complete fusion of cytoplasm has taken place the nuclei unite. The zygote thus formed becomes slightly elongated and develops a cyst wall or oöcyst, which is spindle-shaped. The single nucleus of the zygote, by repeated divisions, gives rise to eight nuclei, which arrange themselves at the equator of the zygote. The zygote then splits longitudinally into eight sporozoites and a central mass of residual cytoplasm, round which the sporozoites lie. It is probable that the first nuclear division in the zygote is a reducing division (Fig. 65).

It is at about this stage that the adult mosquito emerges from the pupal case. The gametocyst ruptures, and the oöcysts are shed into the lumen of the Malpighian tube and make their way to the intestine of the mosquito, from which they escape in the fæces into the water in which newly-hatched larvæ of the mosquito are living. They are eaten by the larvæ, as explained above. The oöcysts are very resistant and capable of withstanding prolonged desiccation. On one occasion eggs of the mosquito collected in West Africa by Dr. Connal were sent to England on dried leaves. These were placed in water and quickly gave rise to a brood of larvæ. It was found by Stevenson and the writer (1915) that the mosquitoes which developed were infected with *L. culicis*. It was evident that the oöcysts had been transported on the dry leaves, and had infected the larvæ hatching from the dried eggs.

SUB-DIVISION OF THE GREGARININA.

As already indicated, the sub-class Gregarinina is divisible into two orders, one of which is a small group (SCHIZOGREGARINIDA), comprising forms which reproduce asexually by schizogony, and the other, a very large group (EUGREGARINIDA), which includes the typical gregarines, which have no method of asexual reproduction, and have a life-cycle very similar to that of *Lankesteria culicis*.

A. *Order:* SCHIZOGREGARINIDA.

The members of this order are chiefly intestinal parasites of arthropods, annelid worms, and tunicates (Fig. 466). They vary very greatly in size,
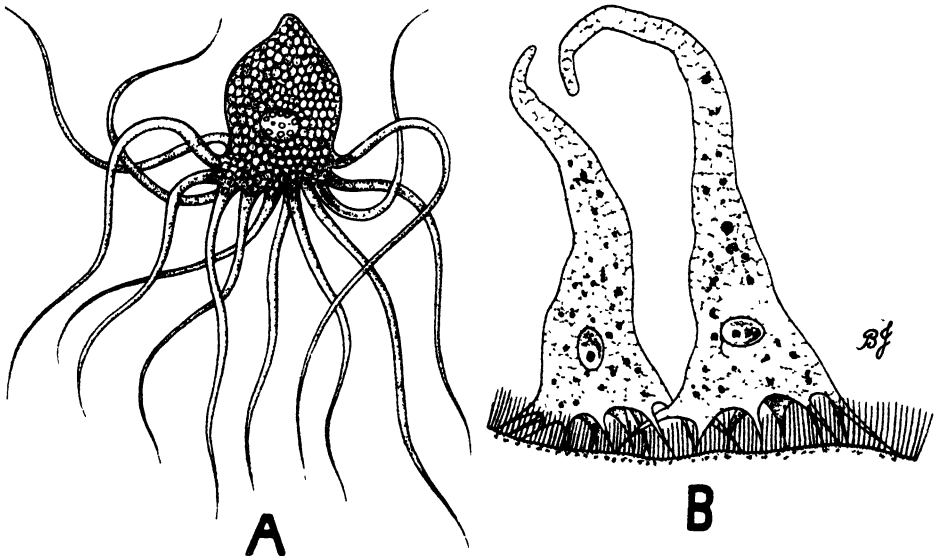
FIG. 466.—SPECIES OF *Ophryocystis*, SHOWING ATTACHMENT PROCESSES (× 2,000). (AFTER LÉGER, 1907.)

A. Uninucleated individual of *O. caulleryi* just after its detachment from the wall of the Malpighian tube of *Scaurus tristis*
B. Two gametocytes of *O. schneideri* attached to epithelium of Malpighian tube of *Blaps magica*.

for members of the genus *Porospora* are amongst the largest gregarines known, reaching a centimetre or more in length, while those of the genus *Lipotropha* are actually the smallest gregarines recorded. In some cases schizogony takes place within cells, while in others it is extracellular. Sporogony is of the typical gregarine type. Two gregarines associate and give rise to gametes, which conjugate to produce zygotes. The latter form oöcysts, each of which contains a number of sporozoites. The

number of oöcysts produced by each pair of associated gregarines varies from one to thirty, while the number of sporozoites in each oöcyst varies from one to eight (Fig. 467).

The Schizogregarinida have been classified in various ways. Léger and Duboscq (1908) divided them into Polysporidea and Monosporidea, according to the number of oöcysts produced by each pair of associated gregarines, while Fantham (1908) recognized two groups, the Ectoschiza
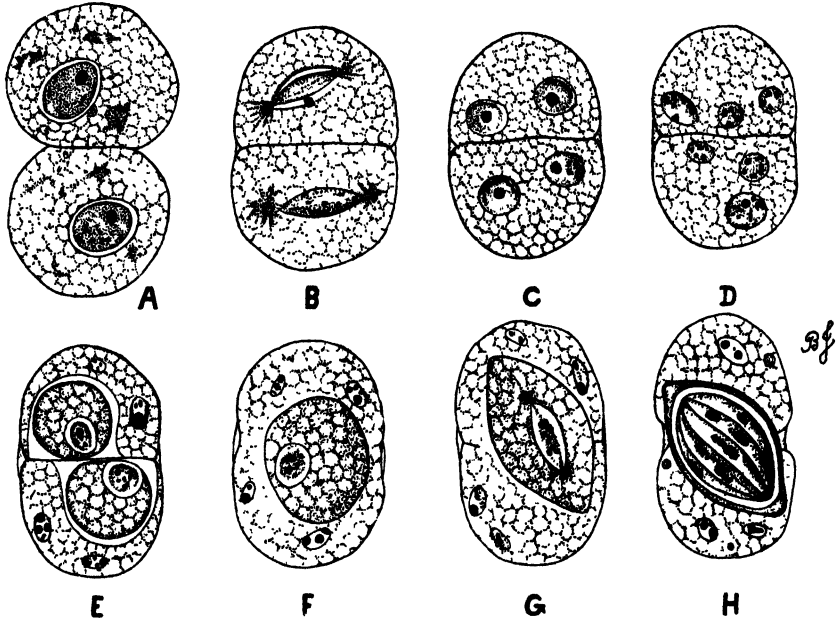
Fig. 467.—*Ophryocystis mesnili* PARASITIC IN MALPIGHIAN TUBES OF THE MEAL-WORM, *Tenebrio molitor* (× 2,000). (AFTER LÉGER, 1907.)

A. Two associated gametocytes.　　　　　　　B. First nuclear division.
C. Gametocytes, each with two nuclei, one of which divides again.
D. Gametocytes, each with three nuclei, two of which degenerate, while one is a gamete nucleus.　　　　　　E. Formation of one gamete in each gametocyte.
F. Zygote resulting from union of gametes.
G. Oöcyst, with included zygote, showing first nuclear division.
H. Mature oöcyst with eight sporozoites.

and Endoschiza, which differ as regards the extracellular or intracellular schizogony. Keilin (1923a) points out that neither of these systems is a natural one, and that it is not impossible that the process of schizogony has been secondarily acquired by different members of the Eugregarinida, and that the various genera of the Schizogregarinida will be distributed ultimately amongst the families of the Eugregarinida. This view was upheld by Mesnil (1899), but Léger, L. (1909), expressed the opinion that the group is a homogeneous one of monophyletic origin. In view of this

uncertainty, Keilin (1923a) does not attempt to subdivide the order, but merely recognizes a number of genera as follows. It must be admitted, however, that it is very doubtful if schizogony occurs in *Porospora* and *Spirocystis*.

Genus: **Ophryocystis** A. Schneider, 1884.—The gregarine phase is represented by conical parasites attached by their bases to the surface of cells by pseudopodial processes. The two associated gregarines produce a single oöcyst containing eight sporozoites. There are nine species which occur in the Malpighian tubes of Coleoptera.

Genus: **Schizocystis** Léger, 1900.—The gregarine phase is vermicular, and two associated gregarines give rise to a large number (about thirty) of oöcysts, each with eight sporozoites. There are two species which are parasitic in the intestine of dipterous larvæ.

Genus: **Caulleryella** Keilin, 1914.—The gregarine phase is represented by ovoid parasites attached to the epithelium. The two associated gregarines give rise to eight oöcysts, each with eight sporozoites. There are four species which are parasitic in the intestine of dipterous larvæ.

Genus: **Lipotropha** Keilin, 1923.—During the whole life-cycle the parasite, which is the smallest known gregarine, is intracellular in the fat body. The active gregarine phase is either completely suppressed or reduced to a short period of life in the perivascular fluid. The two associated gregarines give rise to sixteen oöcysts, each with eight sporozoites. There are two species parasitic in the fat body of diptera.

Genus: **Menzbieria** Bogoyavlensky, 1922.—The parasite is intracellular or extracellular, there being two kinds of schizogony. The two associated gregarines give rise to a large number of oöcysts, each of which has eight sporozoites. There is one species parasitic in the intestine of an acarine (*Hydrachna*).

Genus: **Porospora** A. Schneider, 1875.—The members of this genus are peculiar in that schizogony occurs in decapod Crustacea and sporogony in lamellibranch Mollusca. There are six species.

Genus: **Spirocystis** Léger and Duboscq, 1915.—The single species is parasitic in the body cavity cells, and the growing phase is peculiar in being spirally coiled. Two gregarines do not associate, but those of one type give rise to elongate male gametes, and those of another to ovoid or spherical female gametes. These conjugate, and the zygote gives rise to a single oöcyst containing only one sporozoite. There is one species parasitic in *Lumbricus variegatus*.

Genus: **Selenidium** Giard, 1884.—Schizogony takes place within the intestinal epithelium. The merozoites grow into elongate gregarines, which associate in pairs in the lumen of the intestine. Within each gametocyst are produced a number of oöcysts, each of which contains

four sporozoites. There are several species which are parasitic in marine polychæte worms and *Gephyrea*.

Genus: **Merogregarina** Porter, 1908.—Schizogony occurs within the cells of the intestine. The adult gregarine is attached to the cells by an epimerite. Two gregarines associate and give rise to a single oöcyst, which contains eight sporozoites. There is a single species parasitic in the intestine of an ascidian.

These various genera have been placed in a number of families, but, as Keilin (1923) points out, as each genus requires a separate family it appears quite unnecessary to recognize these at present, as the definitions of such families are merely those of the genera themselves.

SYSTEMATIC DESCRIPTION OF GENERA.

Genus: Ophryocystis A. Schneider, 1884.

The members of this genus are parasites of the Malpighian tubes of beetles (Figs. 466-468). The eight sporozoites liberated from the ingested oöcyst attach themselves to the surface of the cells forming the Malpighian tubes. Here, on the surface of the cell and projecting into the lumen of the tube, each sporozoite grows and becomes a multinucleate adult. It then segments into a number of merozoites, which again attach themselves to the cells and grow into adults, as did the sporozoites in the first instance (Fig. 468, 1-5). After several repetitions of this process there are produced schizonts containing a small number of nuclei, and these segment, not into merozoites, but into forms which will associate in pairs after the manner of the typical gregarines. The products of this last schizogony are therefore gametocytes, like the adult gregarines amongst the Eugregarinida, and associating in pairs they become encysted in gametocysts (Fig. 468, 6-10). Within each gametocyte only three nuclei are produced, and of these only one is used to form the nucleus of a single gamete. Within the gametocyst each gregarine or gametocyte, instead of producing a large number of gametes, produces only one. The two solitary gametes conjugate and become encysted in a spindle-shaped oöcyst, as in the Eugregarinida. Within the oöcyst are formed eight sporozoites. The single oöcyst escapes from the body, and leads to the infection of a new host in the usual manner (Figs. 467 and 468, 11-16).

Genus: Schizocystis Léger, 1900.

This genus was established by Léger, L. (1900*a*, 1909), for a parasite to which he gave the name *Schizocystis gregarinoides*. It is an intestinal parasite of larvæ of the midge, *Ceratopogon solstitialis*. The infection of the gut of the larva is commenced by sporozoites which escape from the ingested oöcysts (Fig. 469). Each sporozoite then attaches itself

to the gut epithelium and develops into an elongate vermicular body, sometimes 150 microns in length, fixed to the gut wall at one end only.   The single nucleus multiplies, and finally by schizogony there are
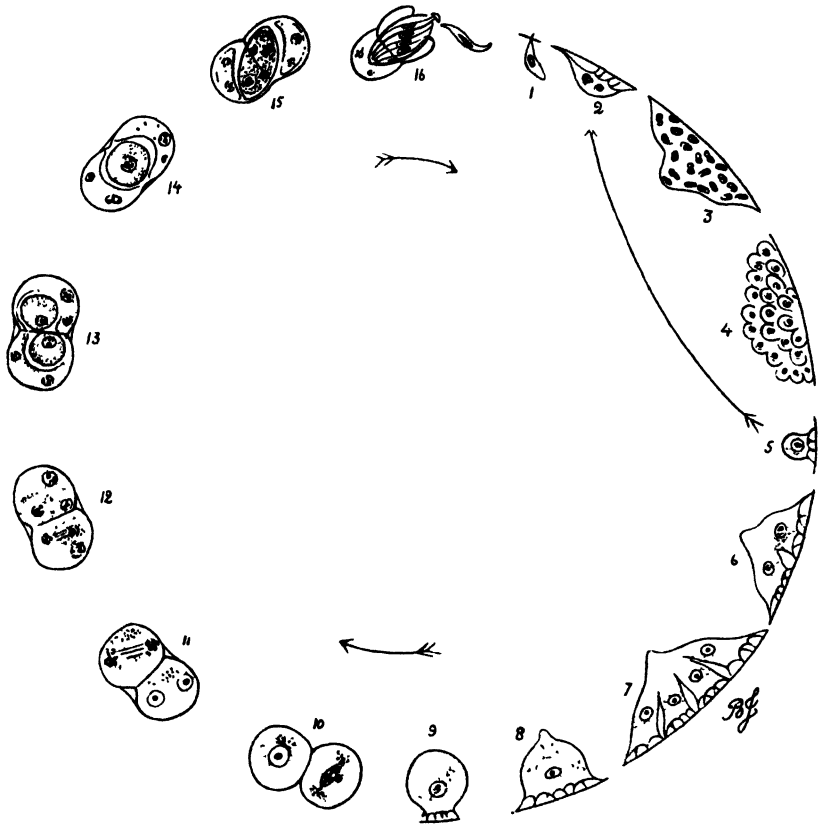
FIG. 468.--DIAGRAM OF LIFE-CYCLE OF *Ophryocystis hessei* PARASITIC IN THE MALPIGHIAN TUBES OF *Omophlus brevicollis* (× *ca.* 1,000).   (AFTER LÉGER, 1907.)

1. Sporozoite attached to epithelium.   2-3. Growing forms with nuclei multiplying.
4. Schizogony into numerous merozoites.
5 Merozoite attached to epithelium.   It may again grow into a schizont of the usual type.
6-7. The merozoite may grow and divide into a small number of gametocytes
8. Single gametocyte attached to epithelium.
9. Gametocyte becoming rounded prior to association with another gametocyte.
10. Association of two gametocytes.
11 Formation of gametocyst round two gametocytes and first nuclear division.
12. One of the two nuclei divides again, forming three nuclei, one of which is the gamete nucleus.
13. Formation of single gamete in each gametocyte.
14. Zygote resulting from union of two gametes.
15. Oöcyst formed round zygote, the nucleus of which has divided to form four nuclei.
16. Mature oöcyst with eight sporozoites, one of which has escaped.

produced numbers of merozoites which again attach themselves to the gut epithelium and become schizonts.   Finally, the merozoites, instead of becoming schizonts, develop without nuclear multiplication into

gametocytes, which, like true gregarines, associate in pairs and become enclosed in gametocysts. Within the cyst, each produces a number of gametes, as in the Eugregarinida. The gametes conjugate in pairs and
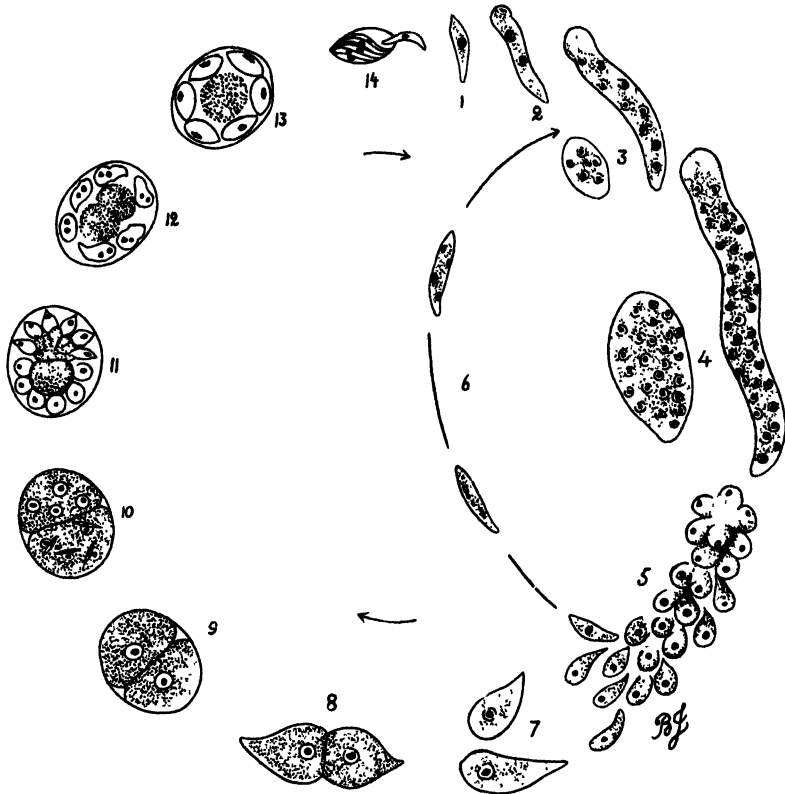
Fig. 469.—Diagram of Life-Cycle of *Schizocystis gregarinoides* Léger, 1909. Parasitic in Intestine of Larvæ of *Ceratopogon* (× *ca.* 1,000). (After Léger, 1909.)

1. Sporozoite escaping from oöcyst in intestine becomes fixed to epithelium.
2-4. Growth into adult schizonts, which are either elongate and attached to epithelium, or globular and unattached. During growth nuclear multiplication takes place.
5. Schizogony with formation of numerous merozoites.
6. Merozoites again become schizonts.   7. Merozoites become gametocytes.
8. Gametocytes associating.   9. Encystment of associated gametocytes in a gametocyst.
10. Nuclear multiplication in gametocytes.
11. Gamete formation; one gametocyte gives rise to spherical female gametes and the other to pointed male gametes.   12. Conjugation of gametes.
13. Zygotes encysted in spindle-shaped oöcysts.   14. Oöcyst with eight sporozoites.

form a number of typical spindle-shaped oöcysts containing eight sporozoites. The oöcysts escape in the fæces, and are taken up accidentally in the food by another host.

Another species which was described by Keilin (1923) as *Schizocystis legeri* occurs in the intestine of *Systenus adpropinquans* and *S. scholtzii*,

which live in decomposing sap or wood pulp in holes in elms and horse-chestnuts. Apart from differences in size of the oöcysts and sporocysts, this form can be distinguished from *Schizocystis gregarinoides* by the fact that the elongate vermicular schizont has a small protomerite separated from a longer deutomerite by a septum.

Genus: **Caulleryella** Keilin, 1914.

This genus was founded by Keilin (1914) for a parasite (*C. aphiochætæ*) which inhabits the intestine of the dipterous larva (*Aphiochæta rufipes*).
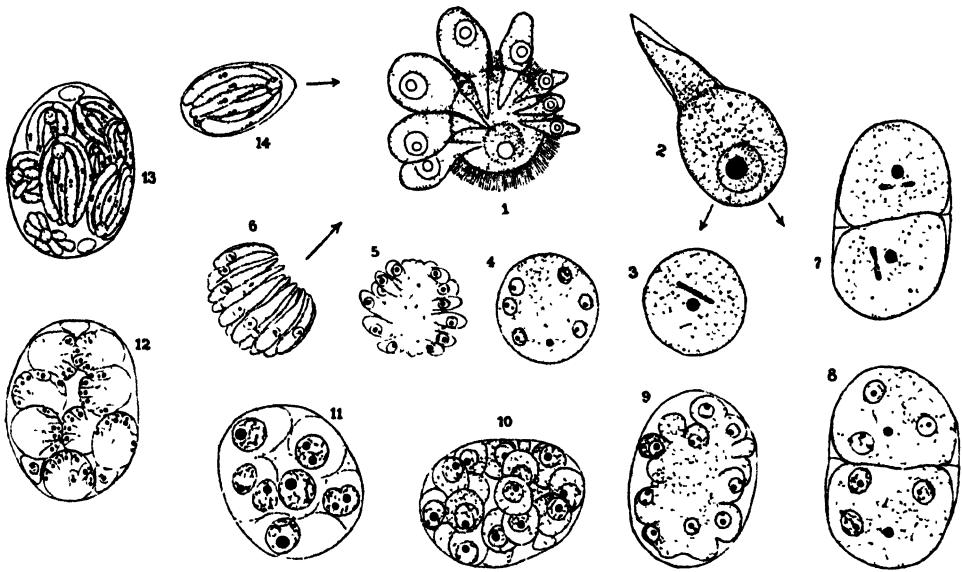
FIG. 470.—LIFE-CYCLE OF *Caulleryella pipientis* PARASITIC IN *Culex pipiens* (× ca. 750). (AFTER BRESLAU AND BUSCHKIEL, 1919.)

1-6. Schizogony cycle. 1 shows an intestinal cell with several attached gregarines.
7-10. Association of two gregarines in gametocyst and formation of gametes.
11. Union of gametes.
12-13. Development of oöcysts and formation of sporozoites.
14. Oöcyst with eight sporozoites.

There are four species which occur in the larvæ of mosquitoes: *C. ano-phelis* in *Anopheles bifurcatus* recorded by Hesse (1918), *C. annulatæ* in *Theobaldia annulata* and *C. pipientis* in *Culex pipiens* by Bresslau and Buschkiel (1919), and *C. maligna* by Godoy and Pinto (1922) in *Cellia allopha* of Brazil.

The life-histories of these parasites are very similar to that of *Schizo-cystis gregarinoides* described above. They differ, however, in that the adult gregarines do not have the elongate vermicular form, but are more or less globular, and possess a process by which they are attached to the

intestinal cells (Fig. 470). The sporozoites liberated from the oöcysts, ingested by the larvæ, escape from the cyst and attach themselves to the cells, in which position they grow into the adult forms. In each of these nuclear multiplication takes place, followed by segmentation into a number of merozoites, which again attach themselves to cells and grow into adults. Finally, two adults associate, withdraw their attachment organs, and surround themselves with a gametocyst. Within this each produces a number of gametes, which conjugate in pairs to produce zygotes. Oöcysts are formed to the number of eight, and within each eight sporozoites are developed. By rupture of the gametocyst, the oöcysts are liberated and escape into the water with the fæces of the larvæ, where they are eaten by other larvæ. When the larvæ pupate the infection dies out, so that the adult mosquitoes are not infected.

Genus: Lipotropha Keilin, 1923.

Of this genus, which was founded by Keilin (1923a), there are two species, both of which are parasitic in the fat body of larvæ of the dipterous genus *Systenus*. They are peculiar in that the whole life-cycle is passed in the cells of this structure, and in being the smallest of known gregarines (Fig. 471). The merozoites become free in the hæmocele fluid, and develop into long vermicules measuring 28 by 1 to 1·2 microns. These associate in pairs in gametocysts, and each gives rise to sixteen gametes. The gametes unite in pairs and the sixteen zygotes become oöcysts, in each of which are eight sporozoites. One species (*L. macrospora*) has oöcysts measuring 13·5 by 3 microns, and the other (*L. microspora*) smaller oöcysts which measure 8 by 3 microns. In infected larvæ the fat body is riddled with parasites in all stages of development.

Genus: Menzbieria Bogoyavlensky, 1922.

The single species (*M. hydrachnæ*) of this genus, which was founded by Bogoyavlensky (1922), is a common parasite of the intestine of an acarine of the genus *Hydrachna*. Reproduction by schizogony is described as taking place in two ways. In the one it is intracellular and clusters of

1. A small portion of the fat body: *a* and *b*, young trophozoites; *c*, more advanced trophozoites; *d*, schizont; *e*, gametocytes; *f*, two associated gregarines; *g*, nuclear multiplication in associated gregarines; *h*, gametocyst with spherical sporoblasts (gametes ?); *i*, gametocysts with more advanced sporoblasts (zygotes ?); *k*, gametocysts with oöcysts; *l*, oöcysts free in the fat body; *N*, nuclei of fat cells.
2-4. Stages in schizogony.   5. Small group of merozoites.
6. Three stages in development of elongate gregarine phase from merozoite.
7-11. Leucocytes of host containing various stages of the parasite: *N*, nucleus of leucocytes; *S*, oöcysts; *p*, various other stages.
12. Abnormal condition of three associated gregarines.
13. Longitudinal section of oöcyst, showing eight nuclei of sporozoites and terminal basophile granules.
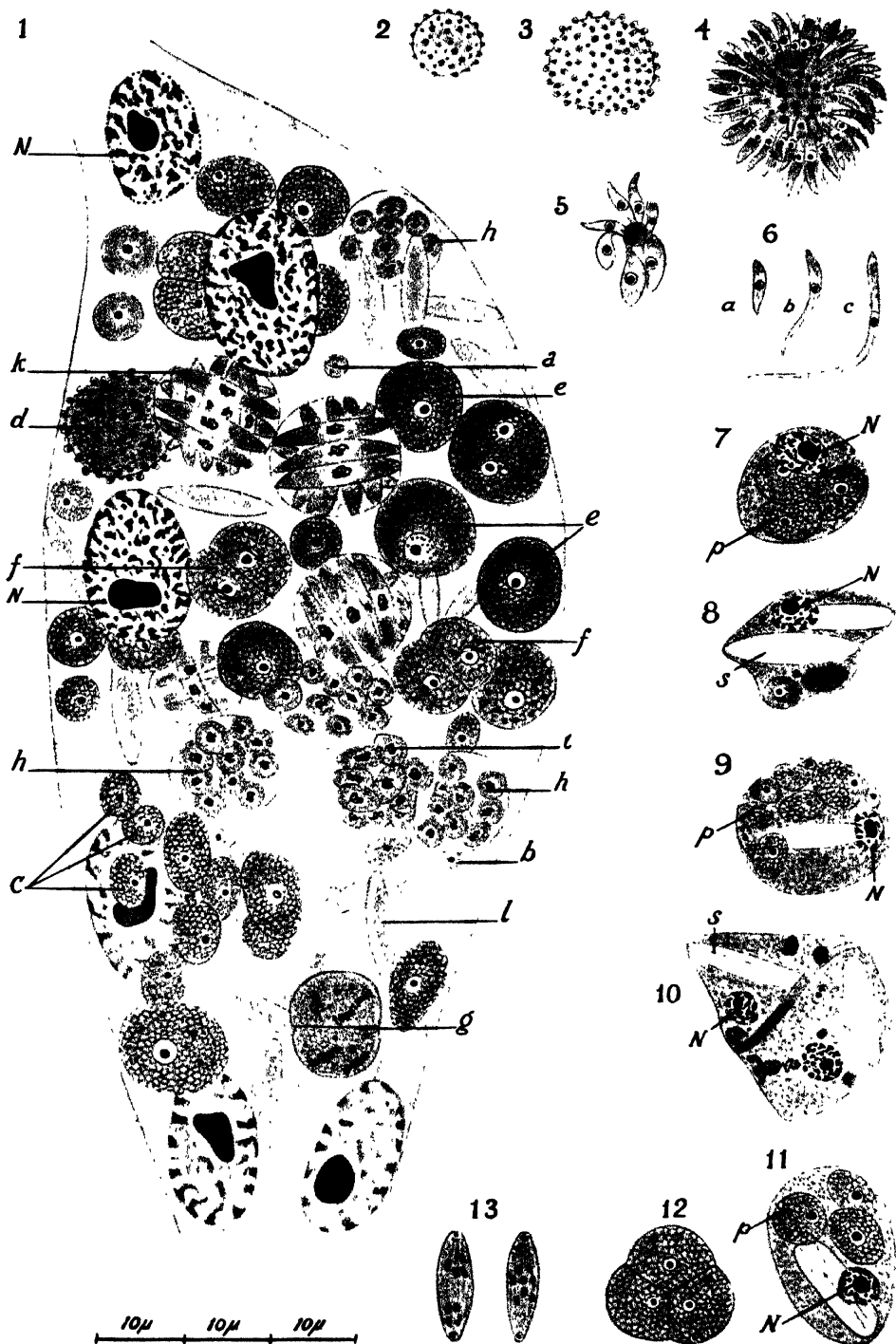
FIG. 471.—DEVELOPMENTAL STAGES OF *Lipotropha macrospora* IN THE FAT BODY
OF LARVÆ OF SPECIES OF *Systenus* (× ca. 1,300). (AFTER KEILIN, 1923.)

elongate merozoites are produced, while in the other it is extracellular and the merozoites are amœboid. It is supposed that the latter have to do with a parthenogenetic process. Sporogony is of the typical gregarine type, two associated gregarines encysting in a gametocyst in which a large number of oöcysts, each with eight sporozoites, is produced.
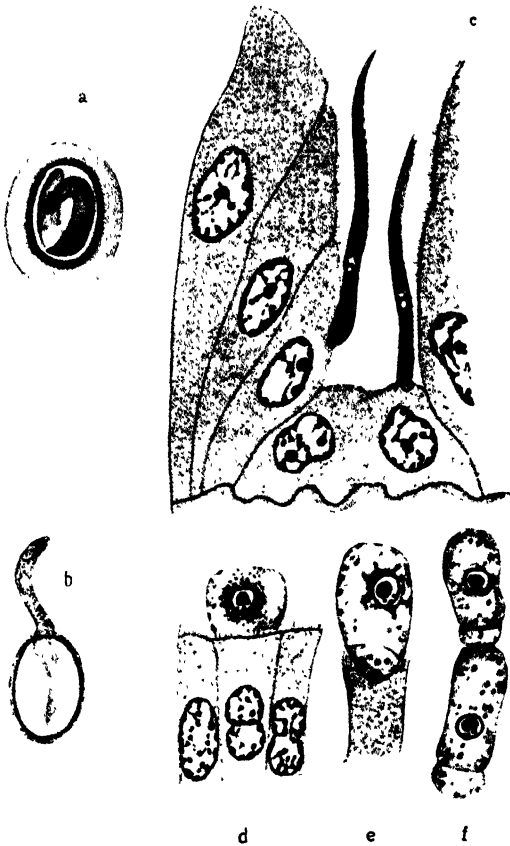
FIG. 472.—*Porospora portunidarum* OF THE SNAIL AND CRAB (× 1,500). (AFTER LÉGER AND DUBOSCQ, 1913.)

   a. Oöcyst containing one sporozoite from the snail, *Cardium edule*.
   b. Escape of sporozoite from oöcyst in intestine of crab.
   c. Sporozoites attached to epithelium of crab's intestine.
d·e. Development of young gregarines.
   f. Two young gregarines in syzygy.
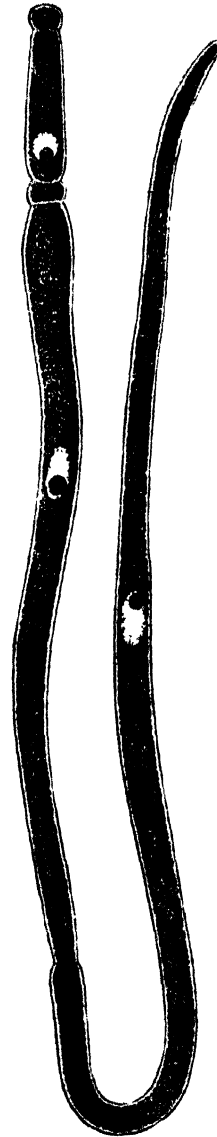
FIG. 473.—*Porospora gigantea* OF THE LOBSTER (× ca. 20): THREE INDIVIDUALS IN SYZYGY. (AFTER LÉGER, 1892.)

Genus: **Porospora** A. Schneider, 1875.

The best-known member of this genus is *P. portunidarum*, which was studied by Léger and Duboscq (1913, 1913a). These observers were able to prove experimentally that an alternation of hosts occurred between crabs of the genus *Portunus* and the mollusc *Cardium edule* (Fig. 472). The mature oöcysts occur in the blood-vessels of the gills of the crab. Each has a thick double wall, and contains a single sporozoite, which eventually escapes through a micropyle when the oöcyst enters the intestine of the crab when it feeds on the mollusc. The escaped merozoite increases in size, and finally attaches itself to the intestinal epithelium, where it develops into a typical large gregarine with definite protomerite and deutomerite. Eventually two gregarines associate and become encysted in a gametocyst. The enclosed gregarines become multinucleated and produce a number of separate cytoplasmic bodies (gymnospore), each with a number of superficially arranged nuclei, which presumably become the gamete nuclei. Conjugation of gametes does not appear to take place in the crab. The gametocysts are passed in the fæces of the crab and are taken up by the molluscs, in which, it is supposed, conjugation occurs and the thick-walled oöcysts are produced.

The life-history of *P. gigantea*, which is a large gregarine found in the intestine of the lobster, appears to be very similar as far as it is known (Fig. 473). Léger and Duboscq (1909b) noted the formation of the gametocysts in the lobster, and the formation of gametes on separate bodies (gymnospores) into which the gregarines segmented. Very frequently a gametocyst was formed round a single gregarine, in which case it may be supposed that the gametes in the cyst are of one sex, and that they will conjugate with gametes produced in another cyst.

Whether reproduction by schizogony takes place or not has yet to be finally determined. If it does not occur, then the genus should be included in the order Eugregarinida.

Genus: **Spirocystis** Léger and Duboscq, 1915.

The single representative of this genus, *S. nidula*, is parasitic in the cells of the body cavity of *Lumbriculus variegatus* (Fig. 474). The growing phase is intracellular, and there is produced an elongate adult which is coiled like a flat spiralled shell. One end of the body is tapering. Nuclear division, followed by the production of numerous merozoites, is described, but it is doubtful if these can again develop into schizonts. It appears probable that they become directly transformed into gametes, those derived from one gregarine becoming narrow, spindle-formed male gametes, and those from another, oval or spherical female gametes. Conjugation

takes place in the body cavity, and the zygote becomes enclosed in a thick-walled oöcyst, within which a single sporozoite is formed. If this cycle has been correctly interpreted, it means that schizogony does not occur, and that a condition has been reached in which the gametocyst is dispensed with, each gregarine, while free, producing gametes which make their way to each other in the body cavity of the host. Like the preceding genus, it is possible that this one should not be grouped with the Schizogregarinida.
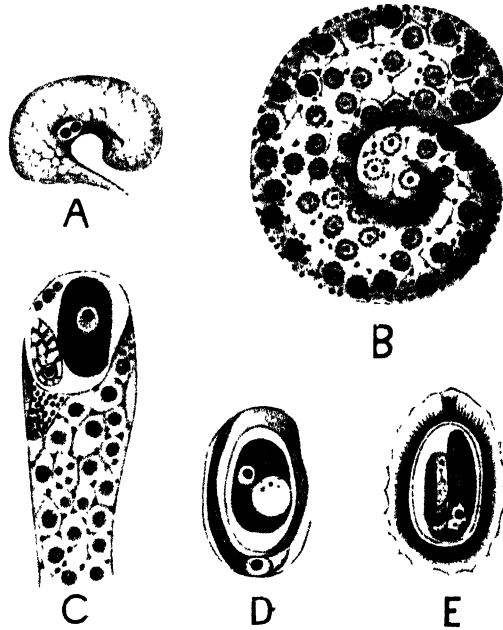
FIG. 474.—*Sporocystis nidula* IN *Lumbriculus*. (AFTER LÉGER AND DUBOSCQ, 1915.)

A. Schizont with one nucleus (× 1,150).
B. Mature multinucleated schizont (× 1,150).
C. Associated microgamete and macrogamete (× 1,150).
D. Possible union of microgamete and macrogamete (× 1,000).
E. Spore (? oöcyst) (× 750).

Genus: **Selenidium** Giard, 1884.

The numerous members of this genus, which was founded by Giard (1884), are chiefly found in the intestine of marine worms. One species occurs in a gephyrean (*Phascolosoma*). The adult gregarines are elongate vermicules, which are often very narrow and provided with conspicuous longitudinal markings. The schizogony of *S. caulleryi* was studied by Brasil (1907) in *Protula tubularia* (Fig. 475). It resembles that of the coccidia, and occurs in the intestinal cells, a large number of sickle-shaped merozoites being produced. The sporogony cycle is not known in
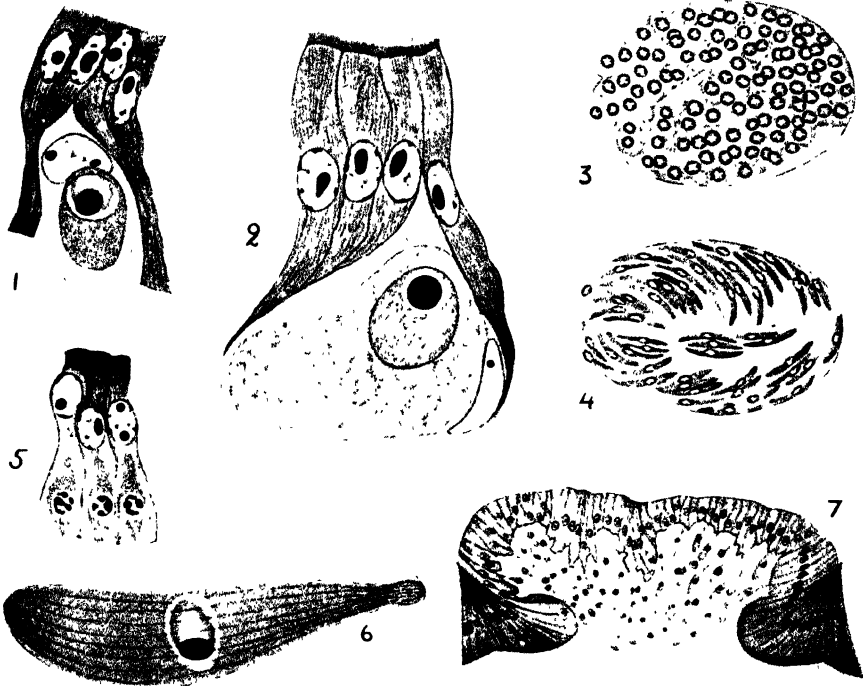
FIG. 475.—STAGES IN DEVELOPMENT OF *Selenidium caulleryi* PARASITIC IN THE INTESTINE OF THE SERPULID, *Protula tubularia.* (AFTER BRASIL, 1907.)

1. Young intracellular schizont (× 620). 2. Older intracellular schizont (× 620).
3. Multinuclear schizont (× 880) 4. Mature schizont with merozoites (× 730).
5. Three young gametocytes in adjacent cells (× 660).
6. Adult gametocyte free in lumen of intestine (× 520).
7. Section of intestinal wall, showing destruction of epithelium by a group of parasites (× 145).
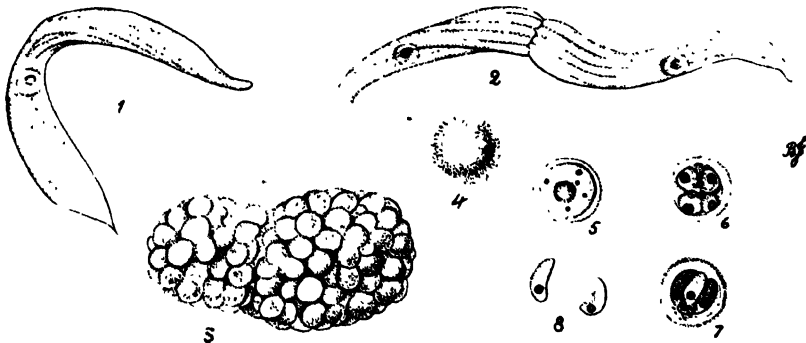
FIG. 476.—STAGES IN THE DEVELOPMENT OF *Selenidium echinatum* IN THE INTESTINE OF THE MARINE WORM, *Dodecaceria concharum.* (AFTER CAULLERY AND MESNIL, 1899.)

1. Free gregarine, showing longitudinal ridges resulting from myonemes (× 300).
2. Two associated gregarines (× 300). 3. Gametocyst filled with oöcysts (× 450).
4. Surface view of an oöcyst (× 850). 5. Oöcyst with undivided zygote (× 850).
6-7. Oöcysts with four sporozoites (× 850). 8. Free sporozoites (× 850).

the majority of species. It was described for *S. echinatum* of *Dodecaceria concharum* by Caullery and Mesnil (1899). Two gregarines associate and form a gametocyst, within which numerous gametes are produced (Fig. 476). These conjugate, and the zygotes so formed become spherical oöcysts, each of which develops four sporozoites. Whether the other species produce oöcysts of this type is not known, for they have only been seen in *S. echinatum*.

<div align="center">Genus: Merogregarina Porter, 1908.</div>

There is but one species, *M. amaroucii* Porter, 1908. It occurs as an intestinal parasite of ascidians of the genus *Amaroucium*. In its cycle of development described by Porter (1908, 1909) it agrees with the species of *Ophryocystis*, from which it differs, however, in that the growing gregarine is an ovoid body attached to the cells by an epimerite. Schizogony is described as taking place within the cells of the gut, so that the gregarine must penetrate the cell before doing this. The oöcyst is of the typical gregarine type, and encloses eight sporozoites, and it is supposed that only a single oöcyst is produced from each pair of associated gregarines.

<div align="center">B. Order: EUGREGARINIDA.</div>

The members of this order have a very uniform developmental cycle, which follows closely that of *Lankesteria culicis*. There is no asexual reproduction, the gregarines developing from the sporozoites being in reality gametocytes, which associate in pairs within a gametocyst. The adult forms are either like those described for *L. culicis*, in which the body is a single unsegmented mass of cytoplasm (**Acephalinidea** Kölliker, 1848), or the body is divided into two portions by a septum of the ectoplasm (**Cephalinidea** Delage, 1896) (Figs. 477 and 478). In the latter case the anterior portion is called the protomerite, and the posterior one, which contains the nucleus, the deutomerite. An attachment organ, the epimerite, which may be elaborately constructed, is present at the anterior end of the protomerite (Figs. 481 and 487).

The subdivision into families of the sub-orders Acephalinidea and Cephalinidea is based on various modifications which involve the shape of the adult gregarines, the character of the epimerite, the type of the gametocysts and oöcysts, and other features, which may be briefly described.

In the Acephalinidea, the sporozoite emerging from the ingested oöcyst penetrates the cell completely, and commences growth as an intracellular parasite. Eventually it becomes too large for the cell which is ruptured, the gregarine then protruding, though remaining attached to the remains of the cell. The majority of the acephaline gregarines are parasites of the body cavity. In the case of the Cephalinidea, the sporozoite, instead of entering the cell, as it frequently does, may become

attached to its surface by its anterior end. As it increases in size it leaves the cell if it has entered it, and develops the complicated epimerite, which may be in the form of a long filament which passes into the cell, a group of anchoring filaments arising from a flattened disc which is applied to the surface of the cell, a kind of rostrum provided
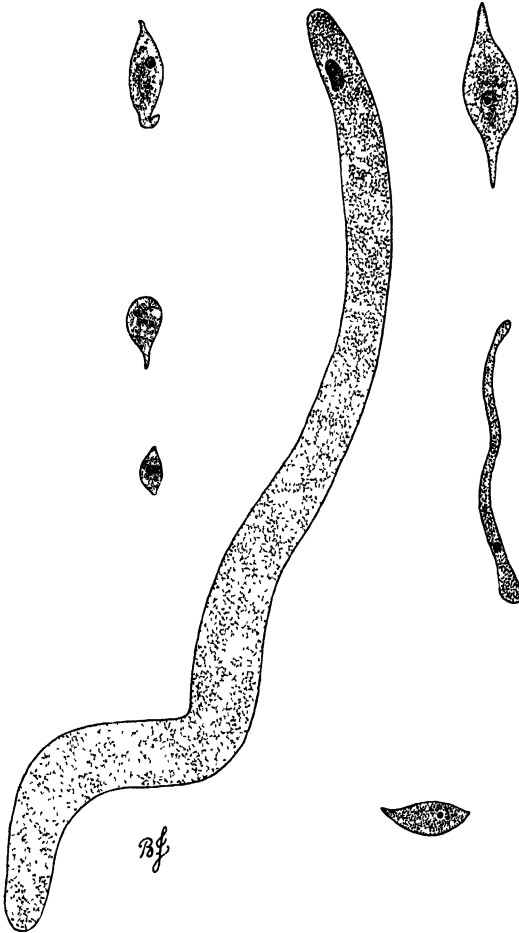
FIG. 477.—ACEPHALINE GREGARINES, *Monocystis agilis* AND *Monocystis magna*, IN FILM OF CONTENTS OF VESICULA SEMINALIS OF EARTH-WORM (× 100). (ORIGINAL.)
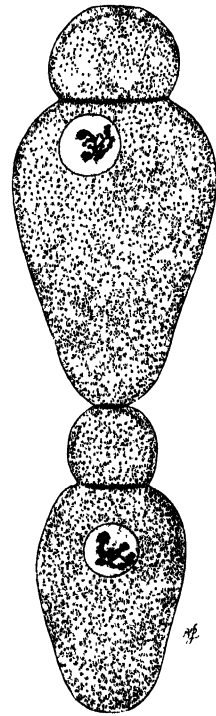
FIG. 478.—CEPHALINE GREGARINE, *Gregarina blattarum*, FROM THE INTESTINE OF THE COCKROACH, SHOWING TWO GREGARINES IN SYZYGY (× 250). (ORIGINAL.)

with recurved hooks not unlike the head of a tape-worm or a sucker. The body in most cases becomes divided by a septum, as described above, into a protomerite and a deutomerite. It is in the latter segment that the large vesicular nucleus lies (Fig. 478). In the family *Doliocystidæ* there

is a simple type of epimerite, and there is no division into protomerite and deutomerite. These forms may be regarded as a connecting link between the cephaline and acephaline gregarines.

Gregarines commencing their growth as sporozoites in an epithelial cell of the intestine, instead of ultimately becoming free gregarines in the lumen of the gut, may pass in an opposite direction and enter the body cavity, where association and encystment occur. In such cases they are spoken of as *cœlomic gregarines* (Fig. 464). In some cases the sporozoites, after liberation from the oöcyst, instead of entering a cell of the gut epithelium, make their way to more distant parts of the body. The common earth-worm is frequently infected with such gregarines (various species of *Monocystis*), the sporozoites of which, after their escape in the
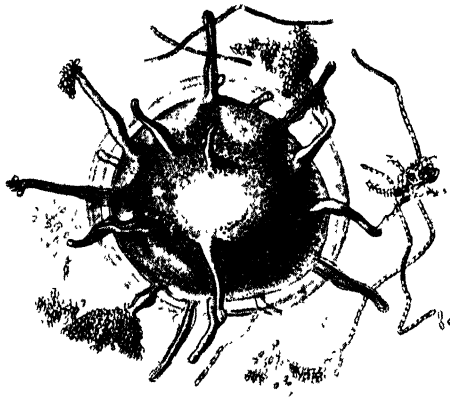
FIG. 479.—GAMETOCYST OF *Gregarina cuneata* OF THE MEAL-WORM, SHOWING OÖCYSTS ESCAPING FROM THE DUCTS (× *ca.* 150). (AFTER KUSCHAKEWITSCH, 1907.)

gut from the oöcyst ingested by the worm, wander to the testis, the sperm mother cells of which are infected (Fig. 484).

Variations occur also in the method of formation of the gametocyst. In many of the cephaline gregarines, special ducts are formed to allow of the escape of the oöcysts. There may be only two or three ducts, or many may be present, and they are formed within the gametocyst and attached to openings in its wall. When they come into operation, they are ejected through the opening like the eversion of an inverted glove finger (Figs. 479 and 480). The oöcysts then pass out of the cyst through these ducts. It appears that the eversion of the ducts and the expulsion of the oöcysts is brought about by the expansion of a large mass of absorbent material which has originated in the residual body derived from the two gregarines after gamete formation. In one case (*Echino-*

mera) the cyst has no ducts, but a large mass lies within the cyst at one side (Fig. 481). When the gametocyst escapes from the body of its host this mass expands, and causes the cyst to rupture and the oöcysts to be scattered. In many cases, however, as in *Lankesteria culicis*, no special expanding body is present, and there are no ducts, the cysts simply rupturing in some unexplained manner after the oöcysts have been formed.

Very interesting modifications occur in the formation of gametes by the two encysted gregarines. In the simplest cases the gametes produced
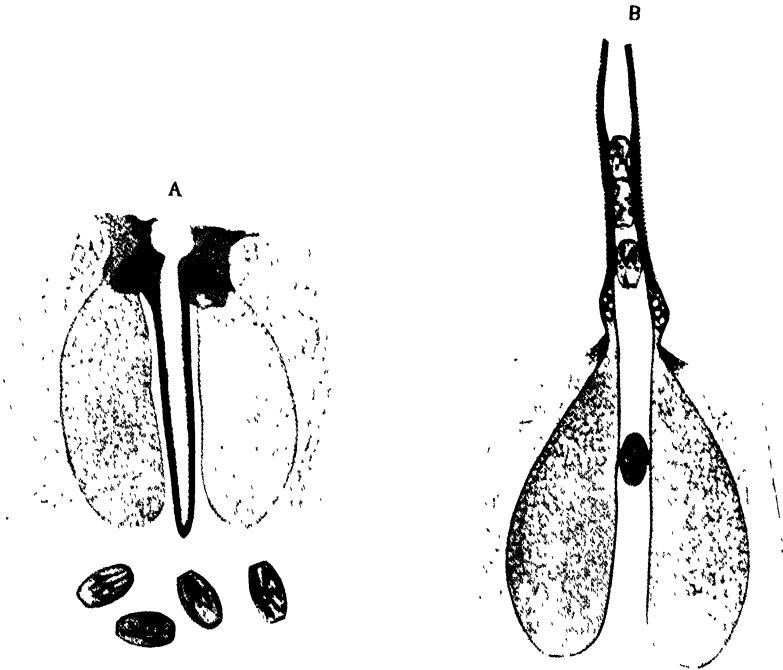
FIG. 480.—TUBULAR DUCTS IN THE GAMETOCYST OF *Clepsidrina ovata* OF THE EARWIG, *Forficula auricularia* (× 850). (AFTER SCHNITZLER, 1905.)

    A. Portion of cyst, showing completely formed duct and four oöcysts.
    B. Everted duct with four oöcysts passing along its lumen.

by such gregarines are not only equal in number, but are indistinguishable in other respects, so that no differentiation into male and female gametes can be made. This is true of some of the gregarines of the common earth-worm, in which there is complete *isogamy*. The first step in a differentiation into male and female gametes occurs in gregarines like *L. culicis*, where the gametes are equal in size, but those produced by one of the gregarines have nuclei larger than those produced by the other. In the case of *Schaudinella henleæ* and *Pterocephalus nobilis* the
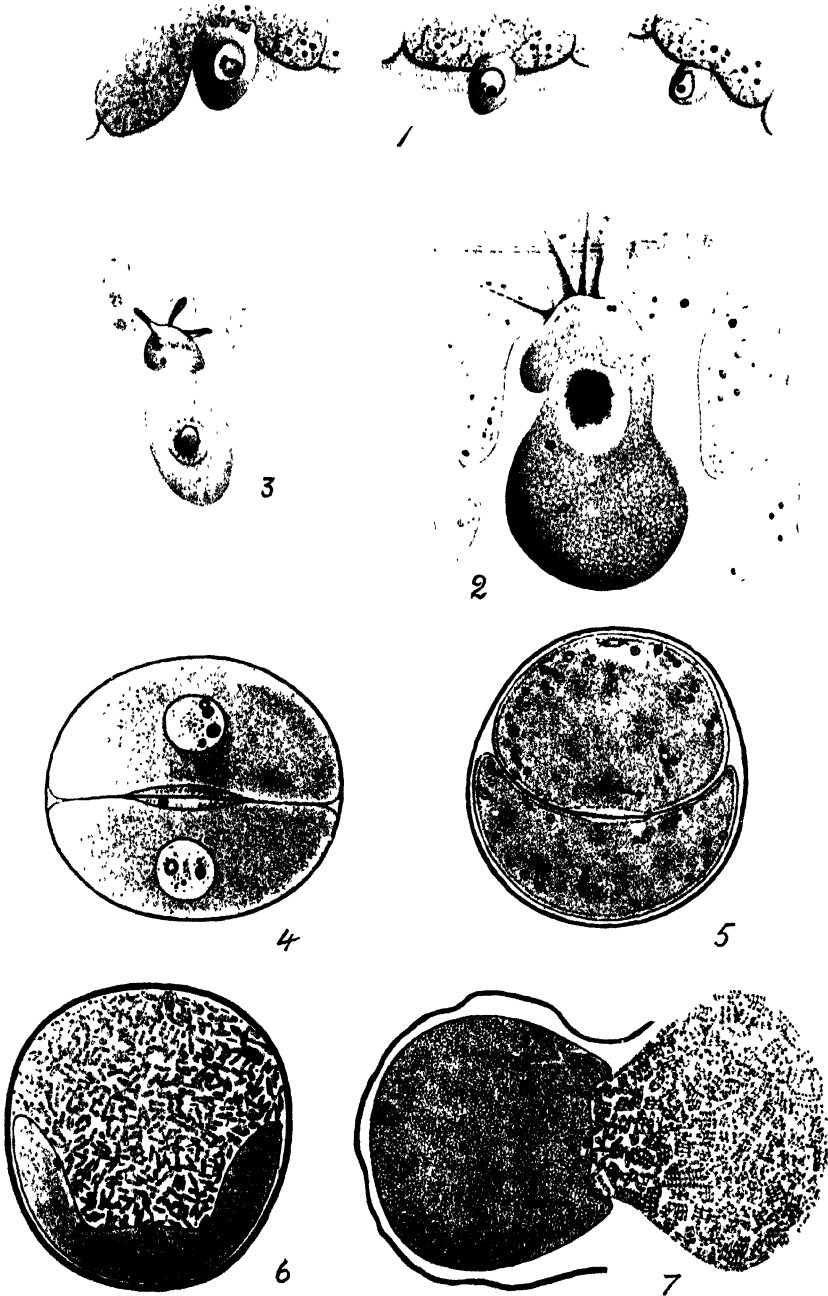
FIG. 481.—*Echinomera hispida* A. SCHNEIDER, PARASITIC IN INTESTINE OF *Lithobius forficatus*. (AFTER SCHELLACK, 1907.)

gametes are definitely unequal in size, producing an anisogamy comparable with that occurring in the coccidia, where the male gamete is so much smaller than the female. In *Stylorhynchus longicollis* and *S. oblongatus*, each of the gametes produced by one gregarine is provided with a flagellum, and is actually larger than those produced by the other gregarine. The latter are spherical bodies, to be regarded as the female gametes, though they are smaller than the male gametes (Fig. 482). It will thus be seen that, as regards the gametes produced in the gametocyst, there is every transition from *homogamy* or *isogamy* (similar gametes) to *heterogamy* or *anisogamy* (dissimilar gametes). In species of *Diplocystis*, one of which was described by Léger and Duboscq (1902) from the body cavity of mosquito larvæ, the two associated gregarines become encysted in the gametocyst, and there occurs a fusion of the cytoplasm. The nuclei, however, remain ununited. Presumably, each nucleus multiplies separately, as in the majority of gregarines, in which no fusion occurs.

In the case of *Lankesteria culicis* it was stated that the gamete nuclei were produced by repeated mitotic divisions after the dissolution of the original nucleus of the gregarine. In some gregarines, another method has been described. Thus Kuschakewitsch (1907), in the case of a gregarine of the meal-worm, described the nucleus as breaking up into a vast number of very minute chromatin granules (chromidia), which are distributed more or less evenly through the cytoplasm. The granules then collect in groups. The granules of each group concentrate more and more till they form nuclei which by divisions give rise to the gamete nuclei. In other cases the chromidial groups are supposed to collect on the surface and then become transformed directly into the gamete nuclei. There is thus a transition from the production of gamete nuclei by repeated divisions from the zygote nucleus to that of direct concentration of chromidia into the gamete nuclei. Recent investigations, however, have not confirmed this method of formation of nuclei from chromidia, and it seems very probable that in all gregarines division takes place by repeated mitoses, as in *L. culicis*. The nucleus of a gregarine is a large structure, within which are one or more large karyosomes. When the nucleus breaks up a spindle is formed, and chromatin in the form of chromosomes appears upon it. The amount of chromatin in the chromosomes

1. Young stages attached to intestinal epithelium, showing development of epimerite ($\times 2,000$).
2. Epimerite fully formed and protomerite developing ($\times 2,000$).
3. Mature gregarine ($\times 550$).
4. Two gregarines freshly encysted in gametocyst ($\times 170$).
5. Nuclear multiplication in the two gregarines, which can be distinguished as a rounded female gametocyte and a flattened male gametocyte ($\times 170$)
6. Later stage in which oöcysts have formed, and the remains of the male gametocyte are represented by a flattened residual body ($\times 170$).
7. The residual body swells and ruptures the gametocyst ($\times 170$).

is very small in comparison with the number of granules formed by the breaking-down nucleus. There is thus a quantity of material, some of which at least seems to be of chromatin nature, which is not used. This has been interpreted as representing a separation of the generative
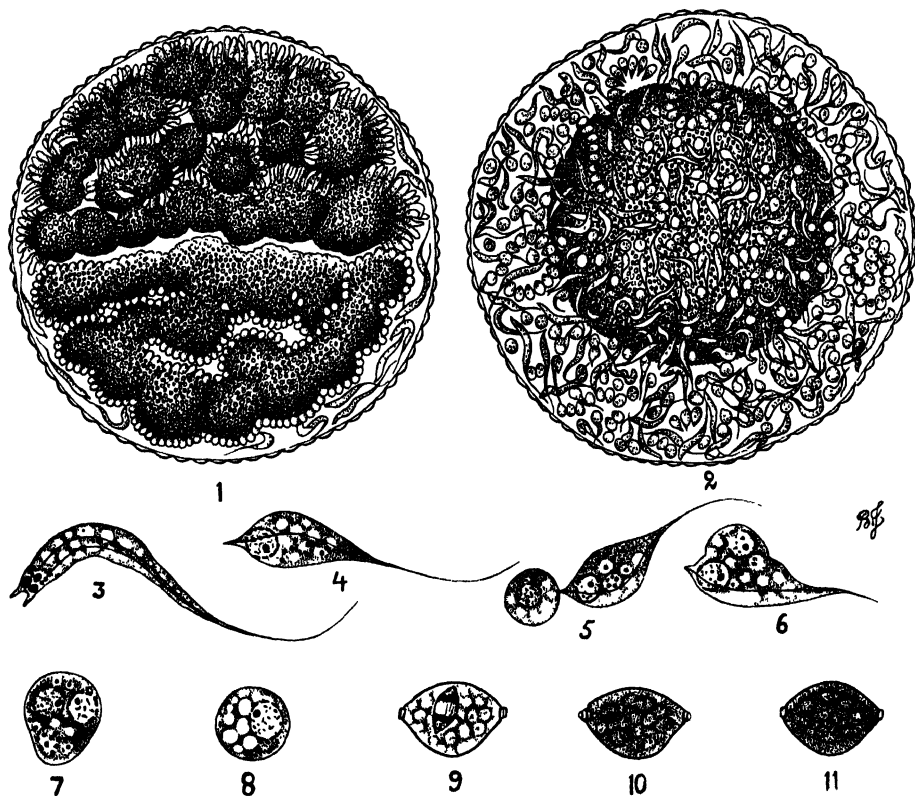
FIG. 482.—SPOROGONY OF *Stylorhynchus*. (AFTER L. LÉGER, 1904, SLIGHTLY MODIFIED.)

1-2. Gametocysts of *Stylorhynchus oblongatus* Hemmerschmidt, 1838, parasitic in intestine of *Olocrates gibbus* Fabr. In 1 the two gregarines (gametocytes) are breaking up into separate masses while budding off gametes, which in the case of one gametocyte are spherical and in the case of the other elongate and flagellated In 2, gamete formation is complete The two types of gamete are commencing to conjugate, and there is a large central residual body (× 170).
3-11. Conjugation of gametes of *Stylorhynchus longicollis* Stein, 1848, parasitic in intestine of *Blaps* sp. and *Scaurus tristis*. 3-4, two types of flagellate gamete; 5-8, stages in conjugation of gametes; 9-11, development of oöcyst (× 1,300).

chromatin in the chromosomes from the vegetative chromatin which is discarded.

Important variations may occur in the oöcysts. In most cases these are spindle-shaped structures which, on account of their resemblance to a well-known diatom, were originally named pseudonavicellæ. Instead

of being spindle-shaped, they may be ovoid, spherical, rectangular, curved, or even provided with filamentous prolongations of their extremities (Fig. 483).

A peculiarity which applies to the cephaline gregarines is a habit they have of attaching themselves to one another when free in the gut (Fig. 478).
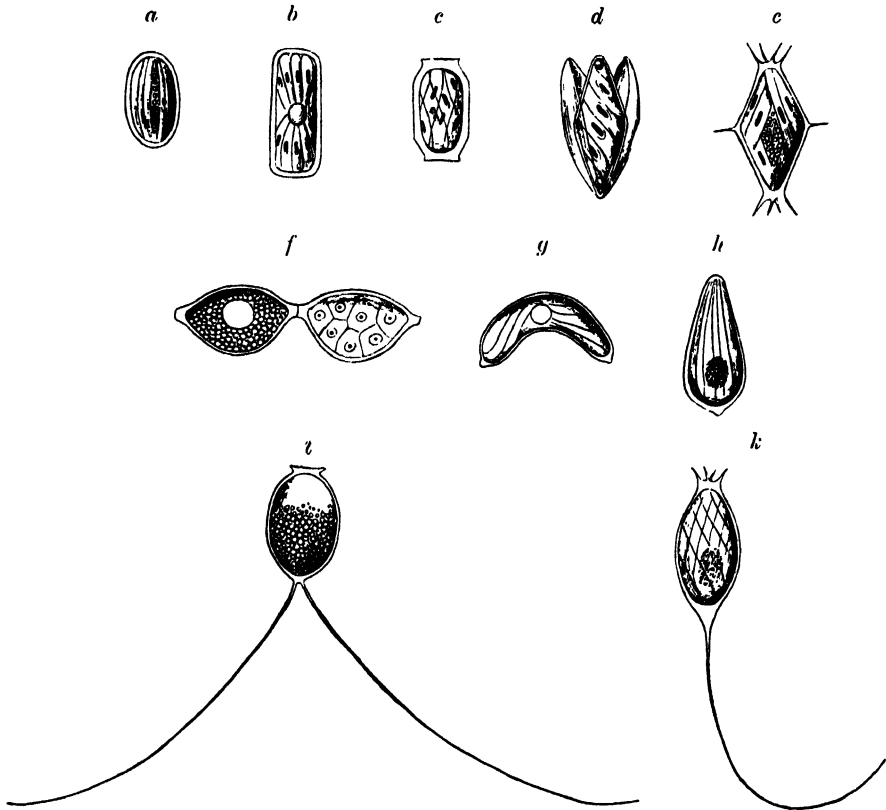
FIG. 483.—VARIOUS TYPES OF OÖCYST PRODUCED BY GREGARINES. (AFTER LÉGER, FROM WASIELEWSKI, 1896.)

a. *Eirmocystis, Sphærocystis,* etc.   b. *Echinomera, Dactylophorus, Pterocephalus,* etc.
c. *Gregarina,* etc.   d. *Beloides.*   e. *Ancyrophora.*
f. *Stylorhynchus.*   g. *Menospora.*   h. *Gonospora.*
i. *Ceratospora.*   j. *Urospora.*

In this manner a chain of many individuals may be formed with the anterior end of one attached to the posterior end of another. Branched arrangements may also be seen. Such an association is termed a *syzygy,* and the group a *syzygium.* It is only of a temporary nature, and must be distinguished from the association which occurs prior to the formation of the gametocyst.
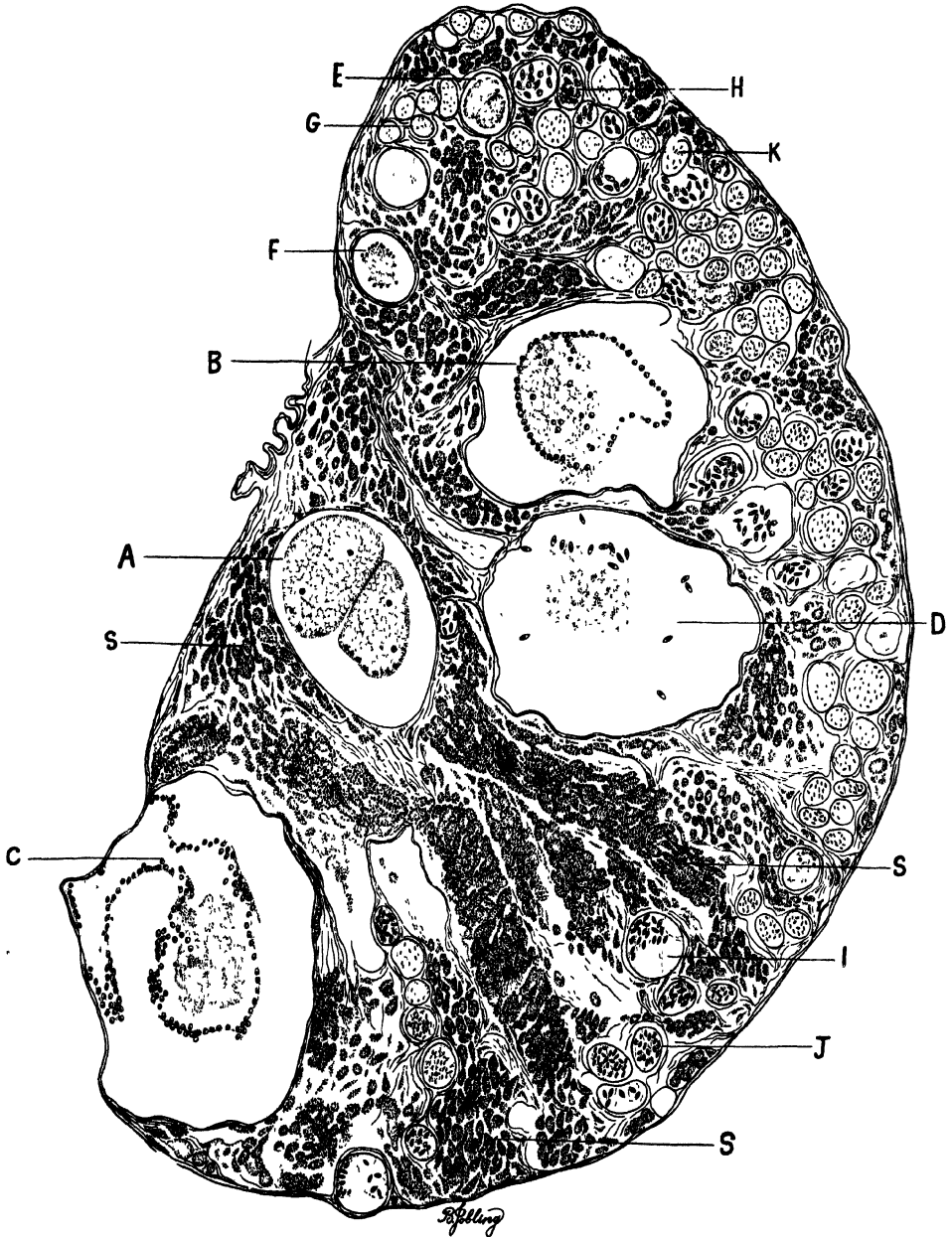
FIG. 484.—SECTION THROUGH A LOBE OF THE VESICULA SEMINALIS OF THE EARTH-
WORM, SHOWING NUMEROUS GAMETOCYSTS OF SPECIES OF *Monocystis* IN
VARIOUS STAGES OF DEVELOPMENT (× 65).   (ORIGINAL.)

The tissue of the organ has been largely replaced by the gametocysts.

[*For description see opposite page.*

Of the acephaline gregarines, those most easily obtained are the various species of *Monocystis* which occur in the testis and vesicula seminalis of earth-worms. They are readily studied in fresh preparations of the teased-out organs or in sections of these (Figs. 477 and 484). The oöcysts ingested by the worm liberate their sporozoites in the intestine, and these make their way to the testes into the sperm mother cells of which they penetrate. Here, in an intracellular position, they increase in size, while the cell (cytophore) continues its development as a multinuclear cytoplasmic body, ultimately producing the spermatogonia and spermatids on its surface. The latter become transformed into spermatozoa, while the host cell by this time has been reduced to a mere membrane surrounding the gregarine. The latter has the appearance of being clothed with cilia, which are the tails of the attached spermatozoa. Eventually, the remains of the host cell rupture and the gregarine escapes. The gregarines then associate in pairs in the vesicula seminalis, form gametocysts, and continue their development in the usual manner.

There appear to be many different species of *Monocystis* in the earth-worms. Some of these are narrow, elongate vermicules reaching a length of nearly a quarter of an inch. Before encysting they retract to a more or less globular form. Other species are smaller than this, and differ from one another in details only (Fig. 477).

Of cephaline gregarines, of which there are at least eight families and over fifty genera, the most readily observed forms are those occurring as intestinal parasites of the common cockroach (*Blatta orientalis*) and the meal-worm (*Tenebrio molitor*). It is probable that each of these hosts harbours more than one species. They are easily observed alive in the liquid contents of the gut, while the details of development can be studied in sections (Figs. 463, 478 and 485). In the case of the gregarines from both these hosts there are well-developed epimerites attaching them to the gut cells. When they detach themselves the epimerite is broken off, so that the free forms are seen with only proto-merites and deutomerites. Syzygy is a common phenomenon, chains of gregarines being frequently seen. When encystment occurs they become globular, and attach themselves to one another by the protomerite. The wall of the gametocyst is a very thick structure, and is perforated in various places. To each of these openings is attached a tube which at

A. Gametocysts of *Monocystis magna* containing two gregarines with multiplying nuclei.
B. Gametocyst in which gamete formation has occurred.
C. Gametocyst after conjugation of gametes.
D. Mature cyst containing oöcysts
E-F. Gametocysts similar to those at A and B, but belonging to a smaller species of gregarine, *M. agilis*.
G-K. Gametocysts containing oöcysts of varying size.
S. Spermatogenesis of the earth-worm.

first is within the cyst wall, but becomes everted after the oöcysts have been formed. By swelling of the body developed from the residual cytoplasm the oöcysts are forced through the tubes, and may actually be observed in living specimens leaving the tubes in the form of a string of highly refractile granules (Figs. 479 and 480).
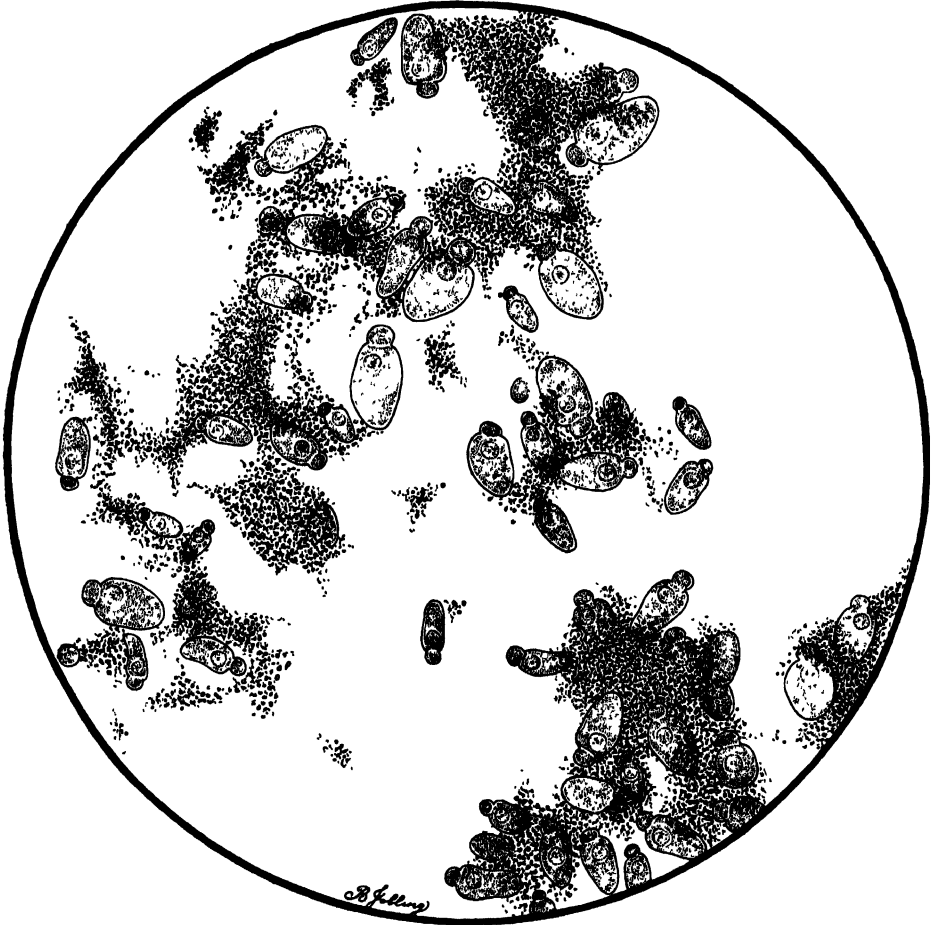
FIG. 485.—*Gregarina blattarum* OF THE COCKROACH.   ONE FIELD OF A SMEAR OF THE INTESTINAL CONTENTS (× 80).   (ORIGINAL.)

The gregarines were free in the lumen of the intestine and had lost their epimerites.

GREGARINES OF BLOOD-SUCKING ARTHROPODA.

In biting arthropods gregarines have been described from time to time, and as these may be encountered in experimental work some reference may be made to them.

Lankesteria culicis of *Aëdes argenteus* has already been described, as also the species of *Caulleryella* found in mosquito larvæ. Günther (1914) described a gregarine of the acephaline type as occurring in the body cavity and respiratory system of the larvæ of *Ficalbia dofleini*, a mosquito of Ceylon.
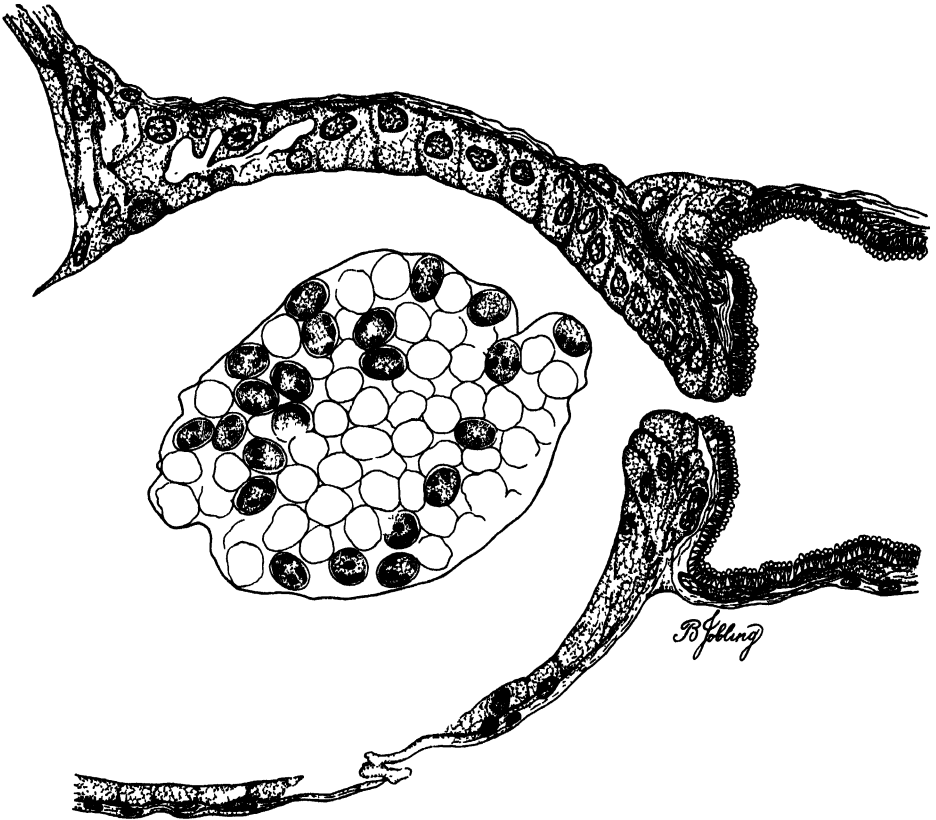
FIG. 486.—LONGITUDINAL SECTION THROUGH POSTERIOR PORTION OF STOMACH. PYLORIC OPENING OF STOMACH, AND ANTERIOR PORTION OF HIND-GUT OF THE DOG FLEA, *Ctenocephalus canis*, SHOWING A GAMETOCYST OF A GREGARINE (*Steinina rotundata?*) WITH NEARLY MATURE OÖCYSTS IN THE STOMACH AND ATTACHED FORMS OF *Leptomonas ctenocephali* ON THE EPITHELIAL LINING OF HIND-GUT (× 700). (ORIGINAL.)

Gregarines have been found in fleas on several occasions. Leuckart (1861) mentions the occurrence of gregarines in the larvæ of fleas. Ross, E. H. (1909), recorded the presence of a cephaline gregarine in the adult dog flea, *Ctenocephalus canis*, in Port Said, and gave it the name *Gregarina ctenocephalus canis*. There was a well-developed epimerite by which the

organism fixed itself to the living cells of the stomach. Characteristic association of two gregarines with the formation of a gametocyst occurred. The oöcysts are described as barrel-shaped. No details of the dimensions are given. What is probably the same form was seen by the writer (1914a) in dog fleas in Malta (Fig. 486). Three large gametocysts almost completely filled the stomach, and in section the oöcysts were seen to be ovoid and to contain eight sporozoites.

The gregarine noted by Nöller (1914) in larvæ of the dog flea is probably the same organism. Wellmer (1910) discovered a gregarine in *Ceratophyllus fringillæ* and *C. gallinæ*. The gregarines possessed epi-
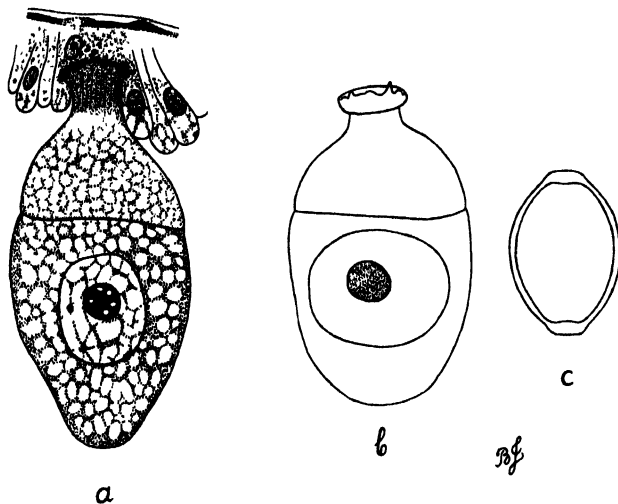
FIG. 487.—*Steinina rotundata* FROM MID-GUT OF *Ceratophyllus styx*, ROTHSCH. OFF SAND-MARTIN. (AFTER ASHWORTH AND RETTIE, 1912.)

a. Gregarine attached to epithelium in section of gut (× 600).
b. Free gregarine (× 600).   c. Oöcyst.

merites provided with eight hooks, while the gametocysts reached a diameter of 180 microns. Wellmer gave it the name *Acanthocephalus parvus*.

Strickland (1912) published an account of a cephaline gregarine seen by him in the larvæ of the rat flea, *Ceratophyllus fasciatus*. The gregarines varied in length from 12 to 55 microns. They were provided with an epimerite consisting of a neck and a disc provided with a ring of finger-like processes. A septum divided the body into protomerite and deutomerite. This gregarine was again studied by Lewin (1913), who gave an account of the encystment which is undoubtedly correct, though differing from that given by Strickland. The gametocysts measure about 75 microns in diameter, and within it oöcysts are produced in the usual

manner.   These are ovoid bodies measuring 7 by 3 microns, and contain eight sporozoites.   To this gregarine Strickland gave the name *Agrippina bona.*

Ashworth and Rettie (1912) described as *Steinina rotundata* a gregarine which they found in three species of flea (*Ceratophyllus styx, C. farreni,* and *C. gallinæ*) obtained from bird-nests (Fig. 487).   It is a typical cephaline gregarine, varying in length from 10 to 180 microns, and showing an epimerite, protomerite, and deutomerite.   The epimerite is in the shape of a disc attached to the body of the gregarine by a neck.   From the centre of the disc a pointed process may extend forwards, while its margins are crenated.   The young stages of the gregarine are attached to the epithelium of the mid-gut by their epimerites, which are embedded in the cytoplasm of the cells.   The gametocysts are spherical, and have a diameter of 110 to 188 microns.   When newly formed, the cyst wall is about 8 microns in thickness, but in older cysts it is thinner than this, so that condensation of the wall must take place.   The oöcysts are spindle-shaped, with the apices of the spindle cut off, so that the ends are flattened.   They are 11 to 12 microns in length by 7 microns in breadth. These oöcysts, when placed in the intestinal fluid of the flea, commence to swell at their apices.   Eventually a kind of plug is detached, exposing a pore through which eight sporozoites may be seen to emerge.   The latter have a length of 9·5 to 10·5 microns and a breadth of 1·5 to 2 microns.

The members of the Kala Azar Commissions working in Assam and Calcutta (see p. 423) have noted that *Phlebotomus argentipes* is liable to be infected with a gregarine, the gametocysts of which occur in the stomach.

RELATION OF THE GREGARININA TO THE COCCIDIOMORPHA.

The chief distinction between the gregarines and the coccidia is that the two gametocytes of the former produce gametes in equal number, and conjugation leads to the production of a similar number of zygotes, whereas, in the latter, one gametocyte produces a number of gametes, while the other, which is invariably the female, produces only one.   In the coccidia, again, the male gametes are small, while the female gamete is as large as the gametocyte itself.   In the case of the gregarines, even when the gametes are sexually differentiated, they are approximately equal in size.   The coccidia always reproduce by schizogony, while with gregarines this method of multiplication occurs only in the small group of the Schizogregarinida, which in this way form a connecting link between the coccidia and gregarines.   In the Eimeriidea the male and female gametocytes develop independently, while in the Adeleidea they are in association, and the condition which occurs in the gregarines is approached.   In the

case of *Adelea ovata* the male gametocyte produces only four male gametes, while in *Karyolysus lacertarum* the number is reduced to two, so that they approach the condition which maintains in the case of the gregarines of the genus *Ophryocystis,* in which each of the associated gametocytes gives rise to only a single gamete.

The gregarines differ from the coccidia in their growing phases. The latter remain as intracellular parasites during the whole of this period, and have no free existence. The gregarines, on the other hand, sooner or later leave the host cell and move about in the gut lumen or body cavity as free motile organisms. In the case of *Selenococcidium intermedium,* on the other hand, the adult form is of the vermicular or gregarine type, and leads a free existence in the gut of its host. Its sexual phase, on the other hand, is that of a coccidium. The gametocytes of *Isospora felis* are definitely gregariniform in character, and may even be attached to the cytoplasm of the cell by their anterior ends (Fig. 347, 19). In the gregarines generally there is a free-living phase, and the two gameto-cytes produce a large number of oöcysts, while in the coccidia there is, as a rule, no free-living phase, and the two gametocytes, which may or may not be associated, produce only a single oöcyst.

B. *SUB-PHYLUM:* CILIOPHORA.

GROUP 1: PROTOCILIATA.

V. *CLASS:* OPALINATA.

CLASSIFICATION.

CLASS: **OPALINATA**
  Family: OPALINIDÆ
    Sub-Family: PROTOOPALININÆ
      Genus: Protoopalina
      ,,   Zelleriella

  Sub-Family: OPALININÆ
    Genus: Cepedea
    ,,   Opalina

In this class there is a single family—OPALINIDÆ Stein, 1860—the members of which it has been usual to regard as belonging to a single genus, *Opalina* Purkinje and Valentin, 1835 (see p. 158). Metcalf (1918, 1920) has, however, established four distinct genera, reserving the genus *Opalina* for forms like the common *Opalina ranarum* of the rectum of the frog. He (1920) recognizes two sub-families—the PROTOOPALININÆ, which includes types with two nuclei; and the OPALININÆ, those with four or more nuclei. The division into genera is based on the shape of the body, which may be more or less cylindrical or flattened and leaf-like. In the sub-family Protoopalininæ there are two genera—*Protoopalina* Metcalf, 1918, the members of which have more or less cylindrical bodies, which are circular or broadly oval in cross-section; and *Zelleriella* Metcalf, 1920, the members of which have more or less flattened bodies which are narrow oval in section. In the sub-family Opalininæ there are, again, two genera - *Cepedea* Metcalf, 1920, and *Opalina* Purkinje and Valentin, 1835. The members of the former genus have more or less cylindrical bodies and those of the latter flattened bodies.

The various species are found chiefly in the intestine of Amphibia, but one, *Protoopalina saturnalis* (Léger and Duboscq, 1904), occurs in a marine fish (*Box boops*). The life-histories have been studied by Neresheimer (1906, 1907), Metcalf (1909, 1923), Brumpt (1915), Konsuloff (1922), and others. In the majority of species the body consists of a thin plate of cytoplasm roughly oval in outline, but some have an elongate cylindrical body, one end of which may be tapering. The whole body is uniformly clothed with longitudinally arranged parallel rows of cilia. There is no cytostome, but some of the elongate forms have an excretory system consisting of a longitudinal canal which communicates with the exterior by

a pore at the posterior end of the body. There is a definite ectoplasm and endoplasm. The latter contains two or more nuclei, and often a number of bodies which are smaller than the nuclei, and have an ovoid or spindle shape. As regards the nature of these bodies there has been much speculation. In many respects they stain like chromatin. Konsuloff (1922) has come to the conclusion that in the case of *Opalina ranarum* they correspond with the macronuclei of other ciliates, and that the structures which have hitherto been regarded as the only nuclei present are in reality the micronuclei. In favour of this view he argues that the spindle bodies appear to arise from material extruded from the nuclei, that they stain like macronuclei of other ciliates, multiply by division, and finally disappear when syngamy occurs. Hickson (1903) came to a conclusion the reverse of this. He considered the spindle bodies to be micronuclei and the nuclei macronuclei. The latter view is evidently incorrect. Metcalf (1923) does not believe in their nuclear nature.

Reproduction is by binary fission. In the binucleate forms the two nuclei divide by mitosis, and when four nuclei are present the body divides into two daughter individuals. In the multinucleate forms nuclear division by mitosis proceeds somewhat irregularly till many nuclei are present. Finally, division of the body takes place, to give rise to two multinucleate individuals.

During nuclear division chromosomes are formed, but in many cases these do not become regularly arranged as an equatorial plate. Nevertheless, it appears that the individual chromosomes actually divide to form those of the daughter nuclei, though at one time Metcalf (1909) was inclined to think that no division of the chromosomes occurred. In his more recent work (1923) he appears to have modified this view. A notable feature of the nuclei, as Metcalf (1923) points out, is a tendency they have of resting after nuclear division has commenced. Cyst formation commonly occurs, and it is by means of cysts that infections spread from one host to another.

Opalina ranarum (Purkinje and Valentin, 1835).—This is the form which has been most extensively studied, as it is nearly always present in the rectum of the common frog, *Rana temporaria*. It can easily be recognized as an actively motile ciliated plate of cytoplasm which has a whitish opalescent appearance (Fig. 488, 5). The individuals vary much in size and shape, and may reach a diameter of nearly a millimetre. They are thus easily seen with the naked eye. The body, which is devoid of cytostome or other opening, is covered by a distinct pellicle, beneath which is the ectoplasmic layer containing bundles of myoneme fibrils and curious vacuoles, in each of which is a granule. The cilia are arranged longitudinally in parallel rows, and are usually supposed to arise from blepharoplasts lying immediately beneath the pellicle. Gatenby and King

(1925, 1926), however, state that the blepharoplasts are in the endoplasm, and that this is an indication that *O. ranarum* is a flagellate. The endoplasm contains numerous nuclei, which are evenly distributed through the cytoplasm. Each is a spherical structure limited by a definite membrane, upon which the chromatin is arranged in the form of granules or as an irregular network. At the centre of the nucleus is a karyosome, which, according to Metcalf (1909), contains no chromatin. The endoplasm also
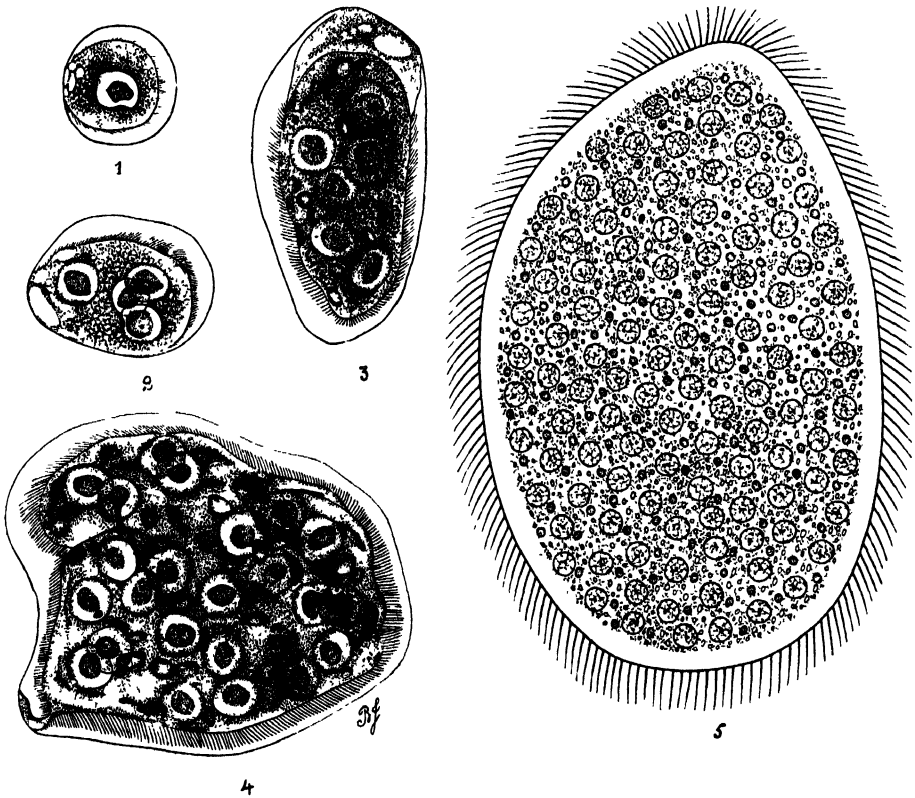
FIG. 488.—*Opalina ranarum* (× 400). (ORIGINAL.)

1-4. Encysted individuals with one or more nuclei. In addition to the nuclei, the cytoplasm contains refringent bodies and deeply-staining granules.   5. Section view of ciliate.

contains the small spindle-shaped bodies referred to above. As the organism increases in size through absorption of nutriment by osmosis, the nuclei multiply by a modified form of mitosis. When a nucleus commences to divide, it increases in size and elongates, and then develops an intranuclear spindle without any trace of centrosomes. The chromatin granules form a number of elongate chromosomes, which become arranged upon the spindle fibres in an irregular manner. Though they do not form a definite

equatorial plate, they divide and become separated into two groups, which move towards the poles of the spindle. The nucleus then constricts at its centre, and two nuclei are formed. According to Konsuloff (1922), the spindle bodies which occur in the endoplasm, and which, he supposes, represent the macronuclei of other ciliates, also multiply by simple constriction (Fig. 489, 23-24), an observation first made by Toenniges (1898).

Multiplication of the ciliate is brought about by division of the body into two parts, each of which is a multinucleate organism. During the greater part of the year the large forms alone are present in the frogs. In the spring, however, repeated divisions of the ciliate give rise to small individuals with only a small number of nuclei. These become enclosed in cysts which measure from 30 to 70 microns in diameter, and are passed into the water by the frogs (Fig. 488, 1-4). The newly-hatched tadpoles ingest these infecting cysts, and the enclosed ciliates are hatched in the rectum. These are gametocytes, which, according to Konsuloff, can be distinguished as male and female (Fig. 489, 4-6). They contain nuclei and degenerating spindle bodies. They divide repeatedly, and give rise to small ciliated individuals with single nuclei. These forms, which are gametes, have elongate bodies with rounded anterior and tapering posterior ends, and may be distinguished as small males and larger females. They are 28 to 30 microns in length, and do not contain the spindle bodies which were present in the earlier stages. Conjugation takes place between the male and female gametes with complete fusion of their bodies and nuclei (Fig. 489, 7-20). According to the descriptions of Metcalf (1909) and Brumpt (1915), the zygotes so formed do not become encysted. Neresheimer (1906, 1907), and more recently Konsuloff (1922), however, claim that the zygote produces a cyst with a diameter of about half that of the infecting cyst which contains a multinucleate gametocyte, and that it escapes in the dejecta of the tadpole, after which it is eaten by a second tadpole. It is then that the zygote emerges from the cyst, and commences to grow into the adult multinucleate individual. According to Konsuloff, under certain adverse conditions entire fully-grown individuals may become enclosed in purely protective cysts (Fig. 489, 21-22). Sometimes the ciliates which emerge from the infecting cysts grow directly into adults instead of producing gametes.

O. ranarum may be regarded as an organism in which division of the cytoplasm does not keep pace with nuclear multiplication, hence the multinucleate condition. In the spring, however, division of the cytoplasm takes place without further nuclear multiplication till the uninucleate gametes are produced. After fusion of the gametes, the zygote so formed increases in size, while nuclear multiplication again proceeds more rapidly than the body divides, and the large multinucleate organisms

result. Whether the spindle bodies are actually macronuclei, as Konsuloff maintains, seems open to question.
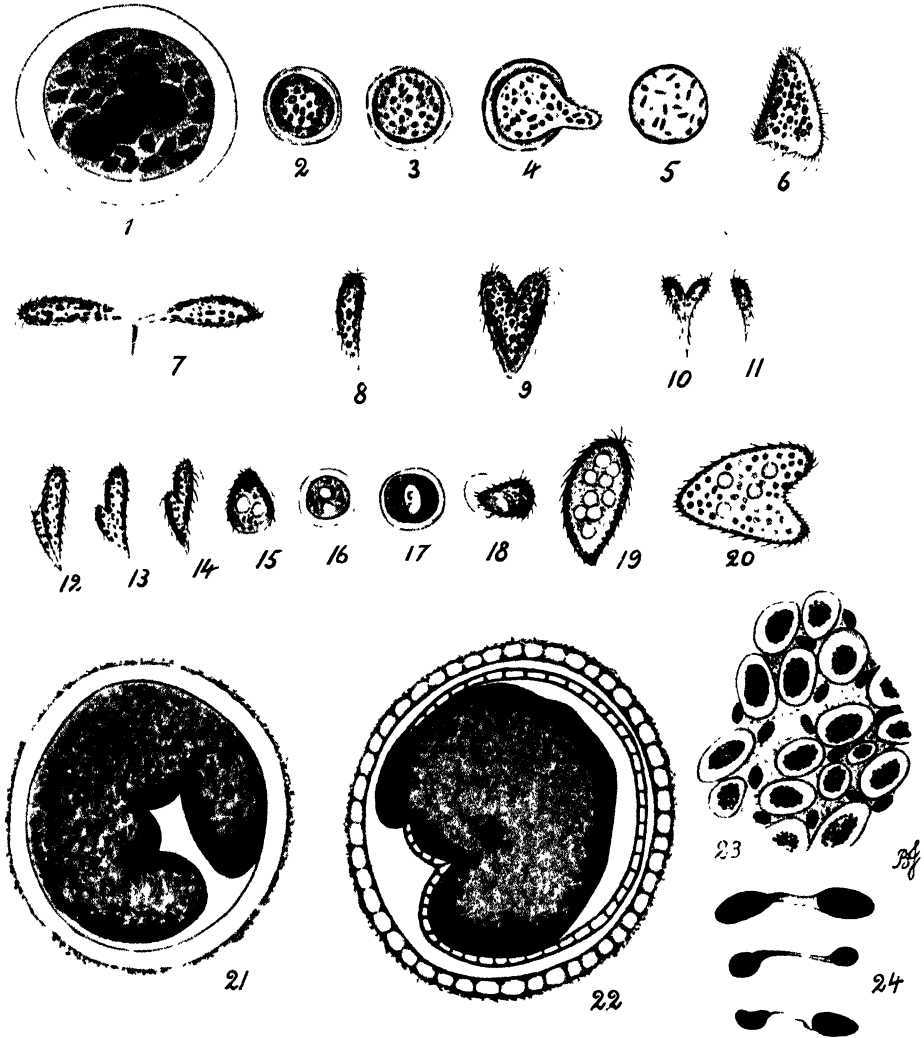
FIG. 489. —*Opalina ranarum :* VARIOUS STAGES IN LIFE-HISTORY (1–23 × *ca.* 500: 24 × *ca.* 1500). (AFTER KONSULOFF, 1922.)

1. Infection cyst containing ciliate with four nuclei and bodies supposed to be macronuclei.
2-6. Escape of ciliate (gametocyte) from infection cyst.
7. Division of macrogametocyte to produce macrogametes.
9-10. Division of microgametocyte to produce microgametes.
12-17. Conjugation of gametes and encystment of zygote.
18-20. Escape of ciliate from cyst and growth to multinucleated form.
21-22. Encysted multinucleate forms (resistant cysts).
23. Endoplasm showing nuclei and bodies interpreted as macronuclei.
24. Division of supposed macronuclei.

8. Macrogamete.
11. Microgamete.

Protoopalina intestinalis (Stein, 1856).—This is a binucleate cylindrical form which occurs in the intestine of various frogs and toads. According to Metcalf (1923), who has given an account of its life-history as far as it is known, it usually varies in length from 180 to 360 microns during the greater part of the year, while in the spring minute forms are gradually produced by more rapid divisions of the body. The ciliate is broad and rounded anteriorly, and tapers to a more pointed end posteriorly (Fig. 490). The body is covered with longitudinal rows of cilia, with a tendency to a spiral arrangement. There is a well-developed ectoplasm consisting of a pellicle, a granular layer in which the basal granules of the cilia occur, and an inner alveolar layer containing certain spherules. The endoplasm contains alveoli, which are smaller than those of the ectoplasm. It also contains numerous bodies, which may be ellipsoidal or dumb-bell-shaped, and numerous small granules. Along the central axis of the body lies
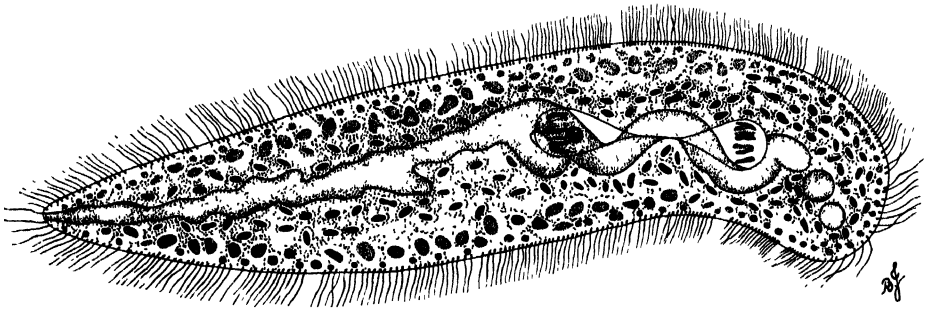
FIG. 490.—*Protoopalina intestinalis* FROM THE INTESTINE OF THE TOAD: SEMI-SCHEMATIC LONGITUDINAL OPTICAL SECTION (× 360). (AFTER METCALF, 1923.)

the excretory organ in the form of an elongate vacuole, which opens posteriorly by a pore. There are two nuclei, which are nearly always pear-shaped and connected by a thread, the remains of the drawn-out membrane of a previous nuclear division. Reproduction is by fission, and this is preceded by mitotic division of the nucleus, during which eight chromosomes are formed. The chromosomes are elongate structures, which are arranged irregularly as an equatorial plate. In addition to these large chromosomes, Metcalf (1923) describes smaller chromatin granules which collect at the equator of the spindle in groups. They lie near the centre of the equatorial plate, and divide into daughter groups, which pass to the poles of the spindle with the larger chromosomes. He regards these as chromosomes of a type differing from the larger ones, and distinguishes macrochromosomes and microchromosomes. The nucleolus of the resting nucleus contains no chromatin, and, without dividing, passes into one or other of the daughter nuclei.

In the spring, by repeated divisions, minute individuals with single nuclei are formed. These encyst and pass into the water, where they are ingested by tadpoles. Here they escape from the cysts and commence dividing, ultimately forming macrogametes and microgametes, which unite in pairs (Fig. 491). The further fate of the zygote has not been traced. It resembles the adult forms except in being smaller and possessing only a single nucleus.
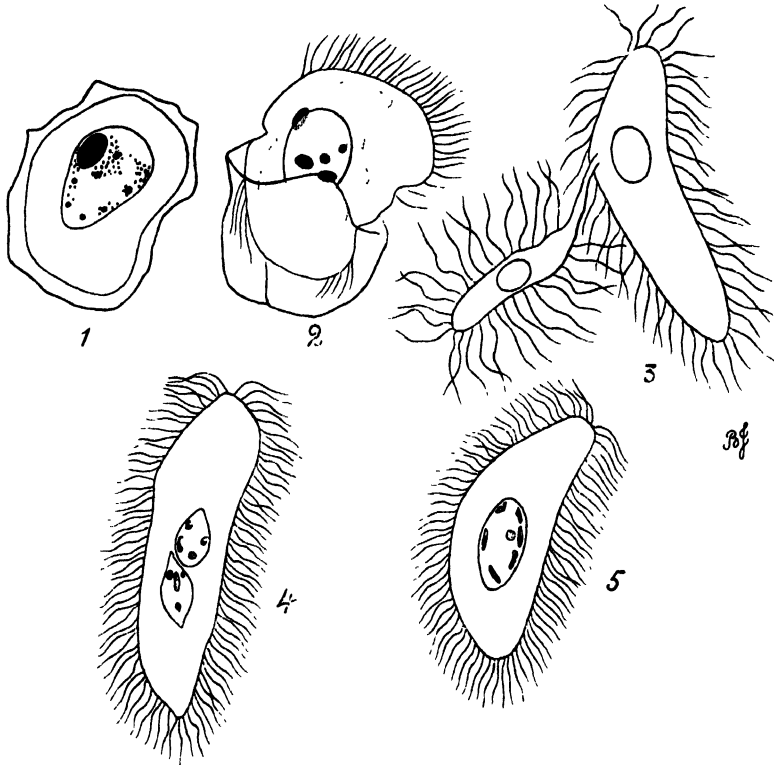
FIG. 491.—ENCYSTED FORMS AND STAGES IN CONJUGATION OF *Protoopalina intestinalis* (× 673). (AFTER METCALF, 1923.)

In the family Opalinidæ there are in all about 150 species which belong to the four genera noted above. They differ from one another in shape, size, and other details. One form, *Cepedea lanceolata*, described by Bezzenberger (1904) from *Rana esculenta* var. *chinensis*, has four nuclei. They are all inhabitants of the intestine of Amphibia, with the exception of the piscine form already mentioned. They do not appear to be in any way pathogenic to their hosts, in which they live as harmless commensals, absorbing nutriment in liquid form from the intestinal lumen.

GROUP 2: EUCILIATA.

VI. *CLASS:* **CILIATA** PERTY, 1852.

CLASSIFICATION.

CLASS: **CILIATA**

  SUB-CLASS: **Aspirigera**
    Order: HOLOTRICHIDA.
      Sub-Order: **Astomatea**
        Family: KOFOIDELLIDÆ
          ,,   INTOSHELLIDÆ
          ,,   ANOPLOPHRYIDÆ
          ,,   DISCOPHRYIDÆ
          ,,   LADIDÆ
          ,,   CEPEDELLIDÆ
          ,,   HERPETOPHRYIDÆ

Family: PEREZELLIDÆ
  ,,   COLLINIIDÆ
  ,,   PROTOPHRYIDÆ
  ,,   ORCHITOPHRYIDÆ
  Sub-Order: **Stomatea**
    Section 1: GYMNOSTOMATA
    Section 2: TRICHOSTOMATA

SUB-CLASS: **Spirigera**
  Order: HETEROTRICHIDA
    ,,   OLIGOTRICHIDA
    ,,   HYPOTRICHIDA
    ,,   PERITRICHIDA

The members of this class are Euciliata which possess cilia at all stages of their development (see p. 158). The shape of the body varies very much in different species. The majority of the Ciliata possess a definite mouth opening or cytostome leading to an œsophagus or cyto-pharynx, which terminates in the endoplasm. Sometimes the mouth opening is situated at the posterior end of a groove or depression, the peristome. A definite anal aperture (cytopyge) is also frequently present at the posterior end of the body, but in those forms with a vestibulum it may be situated in this depression near the cytostome. The numerous modifications undergone by the cilia, such as the fusion of adjacent cilia into stout processes known as cirri, or into membranes in association with the cytostome and cytopharynx, often give the ciliates a very complicated appearance. The cilia are uniformly distributed over the body in longitudinal rows or limited to particular areas. In many forms there is a special zone (adoral zone) of cilia which are arranged in a spiral manner in front of the mouth, and continued into the cytopharynx as cilia or as membranes which have been formed by fusion of adjacent rows of cilia. These forms, as proposed by Blochmann (1886*a*), are known as the **Spirigera** to distinguish them from the **Aspirigera**, which do not possess an adoral zone of cilia. The body may be ovoid, distinctly elongate, or flattened dorso-ventrally. In certain forms the body is more or less cone- or bell-shaped owing to the fact that, after leading a free-swimming existence, they become permanently attached to various objects. The

point of attachment is the apex of the cone, and from it there may be developed a long filament, to the end of which the ciliate is attached. The filament or stalk may be capable of retracting like a spring, and by its means the ciliate is able to draw itself away from objects with which it comes in contact. By repeated divisions of the ciliates at the extremities of the filaments or stalks and the formation of new filaments by the daughter individuals there are produced complicated branched systems, with a ciliate at the extremity of each branch (Fig. 529).

Certain Ciliata are devoid of cytostomes, and in this respect they resemble *Opalina ranarum*, and allied forms. On the basis of this distinction the ciliates devoid of cytostome (including *Opalina ranarum*) have been grouped together as Astomata in distinction to the Stomata, which possess one. This, however,is an unnatural classification in that it involves the grouping of *O. ranarum* with ciliates which have the characteristic nuclear dimorphism.

The names Stomatoda and Astoma were suggested by von Siebold (1848). In the former group he placed the ciliates which possessed cytostomes, and in the latter the ciliate *O. ranarum* and certain flagellates. Later observers removed the flagellates from this group, and included with *O. ranarum* the astomatous ciliates of worms. Ray Lankester (1870) first realized that the grouping of *O. ranarum* with the other astomatous ciliates was an unnatural procedure, and finally Léger and Duboscq (1904a) showed clearly that it differed fundamentally from the ciliates which were usually classed with it. They separated the two types under the headings Opalininæ and Anoplophryinæ. This view was accepted by Cépède (1907, 1910). The important difference between *O. ranarum* and the other ciliates was realized by Marcus Hartog (1906), who went so far as to remove it from the class and place it with the Trichonymphidæ amongst the Mastigophora. The system suggested by Metcalf (1918, 1920, 1923), which is adopted here, appears to be the best, as it recognizes the difference between the nuclei of *O. ranarum* and its allies and those of the remainder of the Ciliophora (see p. 158).

The Ciliata are usually classified according to the distribution of the cilia on the surface of the body, the presence or absence of the cytostome, and the arrangement of the cilia around the cytostome.

CLASS: CILIATA Perty, 1852.

1. *SUB-CLASS:* **Aspirigera** Blochmann, 1886.—When a mouth is present there is no adoral zone of spirally arranged cilia. The body is either uniformly covered with cilia or these are limited to certain areas. The sub-class contains a single order:

(1) *Order:* HOLOTRICHIDA Delage and Hérouard, 1896.—This order

may be subdivided into the forms which are devoid of cytostome (**Astomatea**), and those which possess one (**Stomatea**).

2. *SUB-CLASS:* **Spirigera** Blochmann, 1886.—The peristome has an adoral zone of spirally arranged cilia leading to the cytostome or mouth.

(1) *Order:* HETEROTRICHIDA Delage and Hérouard, 1896.—The adoral zone of cilia is arranged as a left-handed spiral. The body is uniformly covered with cilia of different kinds.

(2) *Order:* OLIGOTRICHIDA.—The general surface of the body is not covered with cilia. The adoral zone is a left-handed spiral.

(3) *Order:* HYPOTRICHIDA Delage and Hérouard, 1896.—The body is flattened dorsoventrally in adaptation to a creeping mode of existence. The cilia are represented mostly by groups of cirri, which on the ventral surface serve for locomotory purposes. There is an adoral zone of cilia arranged as a left-handed spiral in association with the cytostome.

(4) *Order:* PERITRICHIDA Delage and Hérouard, 1896.—The body is cone-shaped and is usually attached to various objects by a stalk which arises from the dorsal surface. It is frequently retractile like a spiral spring. The only cilia present are usually those on the ventral surface which forms in the base of the cone. They are arranged as a right-handed spiral.

The vast majority of the Ciliata are free-living organisms which are found in fresh and salt water. Some of these, such as the various species of *Paramecium* and *Vorticella*, have been extensively studied from the point of view of the phenomena of multiplication and conjugation, and very interesting and important results have been obtained.

Very few ciliates can be regarded as true parasites, though many of them are associated with higher animals, where they live on the surface of the body or in the intestine as commensals.

Many of the stalked or pedunculate forms, such as *Epistylis* (Fig. 529), may become attached to the bodies of aquatic Arthropoda and Crustacea. Such infections may be so heavy, as occurs sometimes in the case of mosquito larvæ, that the hosts appear to be definitely embarrassed. In other cases, free-swimming forms, such as species of *Chilodon* and *Trichodina*, occur in the mucoid material on the skin of fish, while species of *Ichthyophthirius* are true parasites, which actually become embedded in the epidermis. There is a fairly large group of astomatous holotrichous ciliates (Astomatea) which are inhabitants of the intestinal tract, body cavity, and other organs of invertebrates, chiefly worms and Crustacea.

Lamborn (1921) has noted a heavy infection of the body cavity of the larvæ of *Aëdes scutellaris* in the Malay States with a ciliate which Keilin (1921a) named *Lambornella stegomyæ*. A number of other similar infections of insect larvæ has been recorded.

In the intestinal tracts of higher animals ciliates are of frequent occurrence. Frogs and other batrachians harbour species of *Balantidium* and *Nyctotherus*, while both genera are represented in the gut of the cockroach. Ciliates of a very curious and complicated type occur in the stomachs of cattle and other ruminants, and in the cæcum of horses. Closely related forms have been described from the chimpanzee and gorilla and the wild guinea-pigs of Brazil. Finally, there is *Balantidium coli*, which occurs as an intestinal parasite of pigs, and occasionally infects man, giving rise to balantidial dysentery.

Various other ciliates have been described from man, but in most, if not all cases, they are merely coprozoic organisms. Ciliates, like other Protozoa, may become encysted, and in this condition may pass through the intestines and give rise to cultures in fæces after they have left the body. It is possible, but there is no absolute proof of this, that the cysts may occasionally rupture and liberate the ciliates in the lower parts of the intestine, so that the free-living forms are passed in the stool. If this actually occurs, it is in the nature of an accident and must not be regarded as an indication that the ciliates are inhabitants of the intestine.

1. *SUB-CLASS:* **Aspirigera.**

(1) *Order:* HOLOTRICHIDA DELAGE AND HEROUARD, 1896.

In these ciliates the whole body may be covered with a uniform coating of cilia arranged in longitudinal rows, or these may be limited to certain areas. A definite mouth or cytostome may or may not be present (**Stomatea** and **Astomatea**). When it occurs, it is either a simple opening which may lead to a tube or œsophagus ending blindly in the endoplasm, and unprovided with special cilia or membranes (GYMNOSTOMATA Bütschli, 1889), or it may lead to an œsophagus lined with cilia or containing an undulating membrane (TRICHOSTOMATA Bütschli, 1889). Amongst the Holotrichida are usually included a number of well-known free-living ciliates. Such are the various species of *Paramœcium*, *Colpoda*, *Chilodon*, *Prorodon*, *Didinium*, *Lacrymaria*, *Coleps*, *Pleuronema*, *Cyclidium*, *Uronema*, *Glaucoma*, and others. Among parasitic forms are the species of *Ichthyophthirius*, *Anoplophrya*, *Collinia*, *Lambornella* (? *Glaucoma*), and the ciliates which occur in the stomach of cattle and other animals.

The astomatous ciliates (Astomatea) are devoid of cytostome. Their bodies are completely covered by longitudinally arranged parallel rows of cilia. There are numerous families which comprise a number of genera and species.

The Holotrichida (=Holotricha Stein, 1859) may be subdivided as follows:

1. *Sub-Order:* **Astomatea.**—Holotrichida which are devoid of cytostome.

2. *Sub-Order:* **Stomatea.**—Holotrichida which are provided with a cytostome.

Section 1 : GYMNOSTOMATA Bütschli, 1889.—Cytostome usually closed, unprovided with cilia or membranes; œsophagus, if present, naked or supported by rod-apparatus; peristome usually absent.

Section 2 : TRICHOSTOMATA Bütschli, 1889.—Cytostome permanently open, provided with cilia or membranes; œsophagus, if present, ciliated or bearing membranes; peristome usually present.

(1) *Sub-Order:* **Astomatea.**

The ciliates belonging to the Astomatea differ from the Opalinidæ in that each has a macronucleus and micronucleus. They are parasitic chiefly in the intestinal canal and body cavity of invertebrates. Some of them are elongate organisms with long macronuclei, while others are ovoid and possess more rounded macronuclei. In some forms organs of fixation occur at the anterior end of the body, by means of which they can attach themselves to cells, while in others a sucker is developed on the ventral surface of the anterior region of the body. There are usually present several contractile vacuoles, which may be arranged in one or two longitudinal rows. Reproduction is by transverse division of the body. This process may give rise to daughter individuals of equal size, or a small individual may be divided off from the posterior end. By a repetition of this unequal division there may be produced a chain of small ciliates (catenular budding). The process of conjugation has been studied in species of *Collinia.*

Cépède (1910), in his monograph, divides the astomatous ciliates into a number of families and sub-families as follows:

1. *Family:* KOFOIDELLIDÆ Cépède, 1910.
2.   ,,   INTOSHELLIDÆ Cépède, 1910.
3.   ,,   ANOPLOPHRYIDÆ Cépède, 1910.
    (1) *Sub-Family:* RHIZOCARYINÆ Cépède, 1910.
    (2) ,,   ,,   BÜTSCHLIELLINÆ Cépède, 1910.
    (3) ,,   ,,   ANOPLOPHRYINÆ Cépède, 1910.
    (4) ,,   ,,   MESNILELLINÆ Cépède, 1910.
    (5) ,,   ,,   HOPLITOPHRYINÆ Cépède, 1910.
    (6) ,,   ,,   MAUPASELLINÆ Cépède, 1910.
4. *Family:* DISCOPHRYIDÆ Cépède, 1910.
5.   ,,   LADIDÆ Cépède, 1910.
6.   ,,   CEPEDELLIDÆ Cépède, 1910.
7.   ,,   HERPETOPHRYIDÆ Cépède, 1910.
8.   ,,   PEREZELLIDÆ Cépède, 1910.
9.   ,,   COLLINIIDÆ Cépède, 1910.
10.   ,,   PROTOPHRYIDÆ Cépède, 1910.
11.   ,,   ORCHITOPHRYIDÆ Cépède, 1910.

SYSTEMATIC DESCRIPTION OF FAMILIES AND SUB-FAMILIES OF THE ASTOMATEA.

Parasites of the Gastro-Vascular System.

1. *Family:* KOFOIDELLIDÆ Cépède, 1910.

This family contains the single genus *Kofoidella*, of which *K. eleutheriæ* Cépède, 1910, is the sole representative (Fig. 492, 1). It is a pear-shaped organism 30 to 80 microns in length. The body is covered by a delicate membrane uniformly ciliated. The large macronucleus is centrally situated, and a contractile vacuole occurs in the posterior third of the body. According to Cépède, it is a very common parasite of the gastro-vascular system of the medusa, *Eleutheria dichotoma*.

Parasites of the Intestinal Tract.

The majority of the astomatous ciliates are included in this group, in which Cépède recognizes four families, one of which, the Anoplophryidæ, is divisible into six sub-families. The ciliates occur in the intestine of a large variety of hosts, most of which are aquatic worms. They are also found in Rotifera, Bryozoa, Gastropoda, and Batrachia.

2. *Family:* INTOSHELLIDÆ Cépède, 1910.

This family includes the single genus *Intoshellina*, with one species, *I. maupasi* Cépède, 1910, from the intestine of *Tubifex* sp. Like the members of the next family, it has an elongate body covered by parallel rows of cilia which have a longitudinal but slightly spiral course (Fig. 492, 2). There is an elongate macronucleus, and five to seven contractile vacuoles. The characters which justify its inclusion in a separate family are the presence at the anterior end of the body of an organ of fixation and a rudimentary œsophagus, a vestige of the cytostome and œsophagus which were presumably present in the free-living ancestor.

3. *Family:* ANOPLOPHRYIDÆ Cépède, 1910.

Cépède, as noted above, divides the family into six sub-families, the members of which resemble one another in the elongate body which is uniformly ciliated, the well-marked ectoplasm, the numerous contractile vacuoles, the elongate macronucleus, and the absence of cytostome or œsophagus. They differ in the character of the macronucleus, which is cylindrical in all except the Rhizocaryinæ, in which it has lateral branches; in the possession of an armature for fixation purposes, which is present in the three sub-families Mesnilellinæ, Hoplitophryinæ, and Maupasellinæ, and absent in the others; and in the arrangement of the contractile vacuoles and other details.
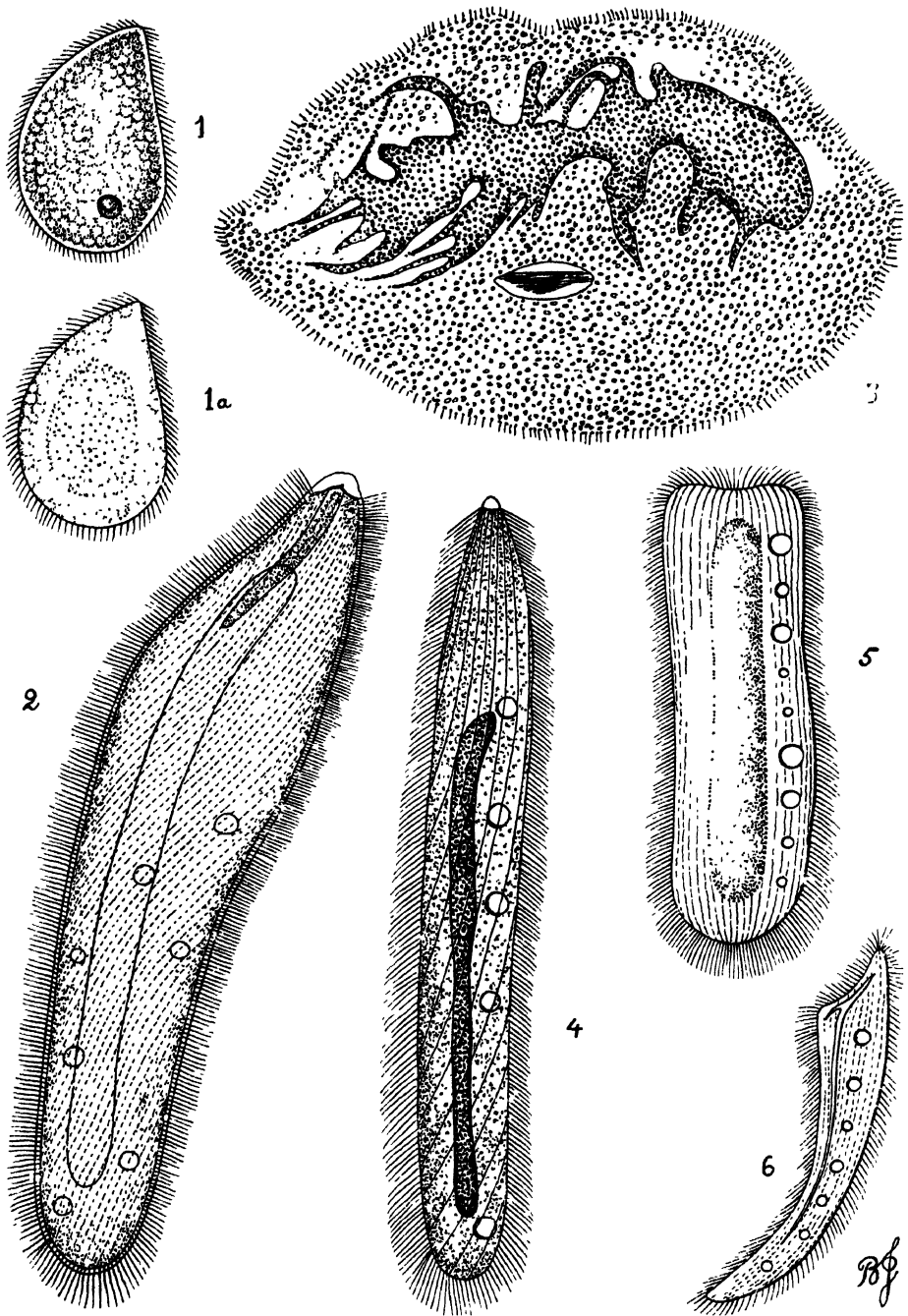
FIG. 492.—VARIOUS ASTOMATEA. (FROM CÉPÈDE, 1910.)

(1) *Sub-Family :* RHIZOCARYINÆ Cépède, 1910.

In this sub-family there is a single genus, *Rhizocaryum*, of which there is but one species, *R. concavum*, Caullery and Mesnil, 1907 (Fig. 492, 3). It is an ovoid organism with a striated depression on the ventral surface. Its chief characteristic is the presence of an elongate branched macronucleus. It is an intestinal parasite of the marine worms *Polydora cœca* and *P. flava*. In an individual 110 microns in length the macronucleus measured 85 microns.

(2) *Sub-Family :* BÜTSCHLIELLINÆ Cépède, 1910.

This sub-family contains a single genus and species, *Bütschliella opheliæ* Awerinzew, 1907. The ciliate measures 280 to 360 microns in length and 25 to 50 microns in breadth (Fig. 492, 4). The body is uniformly ciliated save for a cone of cytoplasm at the anterior end, which may be invaginated. There is a row of about six contractile vacuoles along one side of the elongate macronucleus. Reproduction takes place by the separation of small daughter individuals, which may remain attached in chains (catenular budding). The ciliate is parasitic in the marine worm, *Ophelia limacina*.

(3) *Sub-Family :* ANOPLOPHRYINÆ Cépède, 1910.

The members of this sub-family, which belong to the single genus *Anoplophrya*, resemble those of the preceding sub-family except that the body is uniformly ciliated, there being no anterior cone free from cilia (Fig. 492, 5). The ciliates are elongate organisms which are either cylindrical or slightly flattened. The extremities are rounded, the anterior being often broader than the posterior. Reproduction is by transverse division which may lead to the separation of chains of buds from the posterior end of the body.

The members of the genus occur in the intestine of Polychæta (*Nais serpentina*), and Oligochæta (*Lumbricus terrestris*), leeches (*Clepsine binoculata*), Gasteropoda (*Paludina decisa*), Rotifera (*Noteus*), Bryozoa, and Crustacea (*Homarus gammarus*).

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1. *Kofoidella eleutheriæ* Cépède, 1910 (× 400), from the gastric system of the cœlenterate, *Eleutheria dichotoma*, as seen in living condition (1) and after treatment with alcohol (1a).
2. *Intoshellina maupasi* Cépède, 1910 (× 420), from intestine of *Tubifex* sp., showing elongate macronucleus, contractile vacuoles, and fixation organ.
3. *Rhizocaryum concavum* Caullery and Mesnil, 1905 (× 1,000), from *Polydora cœca* and *P. flava*, showing branched macronucleus and dividing micronucleus.
4. *Bütschliella opheliæ* Awerinzew, 1907 (× 300), from intestine of *Ophelia limacina*, showing macronucleus, contractile vacuoles, and non-ciliated anterior extremity.
5. *Anoplophrya naidos* (Dujardin, 1841) (× 400), from intestine of the polychæte worm, *Nais serpentina*, showing uniformly ciliated body, macronucleus, and contractile vacuoles.
6. *Mesnilella clavata* (Leidy, 1855) (× 300), from intestine of *Lumbricus variegatus*, showing contractile vacuoles and internal spicule.

(4) *Sub-Family :* MESNILELLINÆ Cépède, 1910.

Like the preceding sub-family, the present one contains a single genus, *Mesnilella*, the members of which resemble closely those of the genus *Anoplophyra*, from which, however, they can be distinguished by the presence of a longitudinal rod, which appears to function as a supporting structure. *M. secans*, which may reach a length of 240 microns, with a breadth of 38 microns, occurs in the intestine of the worms *Lumbricus terrestris* and *Lumbriculus variegatus*. The latter also harbours another species, *M. clavata*, which was discovered by Leidy (1855) (Fig. 492, 6).

(5) *Sub-Family :* HOPLITOPHRYINÆ Cépède, 1910.

The ciliates of this sub-family, of which there is the single genus *Hoplitophrya*, possess at the anterior end of the body a chitinous organ of fixation, by means of which attachment to the intestinal wall can be effected. The species *H. lumbrici* (Dujardin, 1841) occurs in the intestine of the earth-worm, *Lumbricus terrestris* (Fig. 493, 1). The organ of attachment is embedded in the cytoplasm, and can be protruded through the anterior end of the body.

(6) *Sub-Family :* MAUPASELLINÆ Cépède, 1910.

In this sub-family there are two genera—*Maupasella* Cépède, 1910, and *Schultzellina* Cépède, 1910—the members of which agree with one another in the possession of a fixing organ at the anterior end of the body in the form of a pointed rostrum, from the base of which myoneme fibres radiate into the cytoplasm.

Maupasella nova Cépède, 1910, is a parasite of earth-worms (*Lumbricus*) of Algiers. It occurs in two forms (Fig. 493, 2). There are ovoid individuals which are broadest in front, and measure up to 82 by 39 microns, and also long, narrow forms up to 117 by 18 microns.

1. *Hoplitophrya lumbrici* (Dujardin, 1841) (× 200), from intestine of *Lumbricus terrestris*, showing micro- and macro-nucleus, fixation organs, and rows of contractile vacuoles.
2-2a. *Maupasella nova* Cépède, 1910 (× 420), from intestine of *Lumbricus* sp., showing micro- and macronuclei, fixation organ, and contractile vacuoles.
3. *Schultzellina mucronata* Cépède, 1910 (× 1,000), from intestine of *Allurus tetrædurus*, showing micro- and macro-nucleus and fixation organ.
3a. Fixation organ on larger scale.
4. *Lachmannella recurva* (Clap. and Lach., 1858) (× 150), from intestine of *Planaria limacina*, showing nucleus, contractile canal, and fixation organ.
5. *Steinella uncinata* (Schultze, 1851) (× 300), from intestine of *Planaria ulvæ*, showing macronucleus, contractile canal, and fixation organ.
6-6a. *Discophrya planariarum* (Siebold, 1839) (× 100), from intestine of *Planaria torva*, showing macronucleus, contractile canal, and terminal sucker.
7. *Haptophrya gigantea* (Maupas, 1879) (× 85), from intestine of batrachians, showing macronucleus, contractile canal, and sucker.
7a. *H. gigantea* reproducing by catenular budding.
8. *Lada wrzesniowskii* Vejdovsky, 1882, from the oligochæte worm, *Pheatothrix pragensis*, showing macronucleus, contractile vacuole, and sucker.
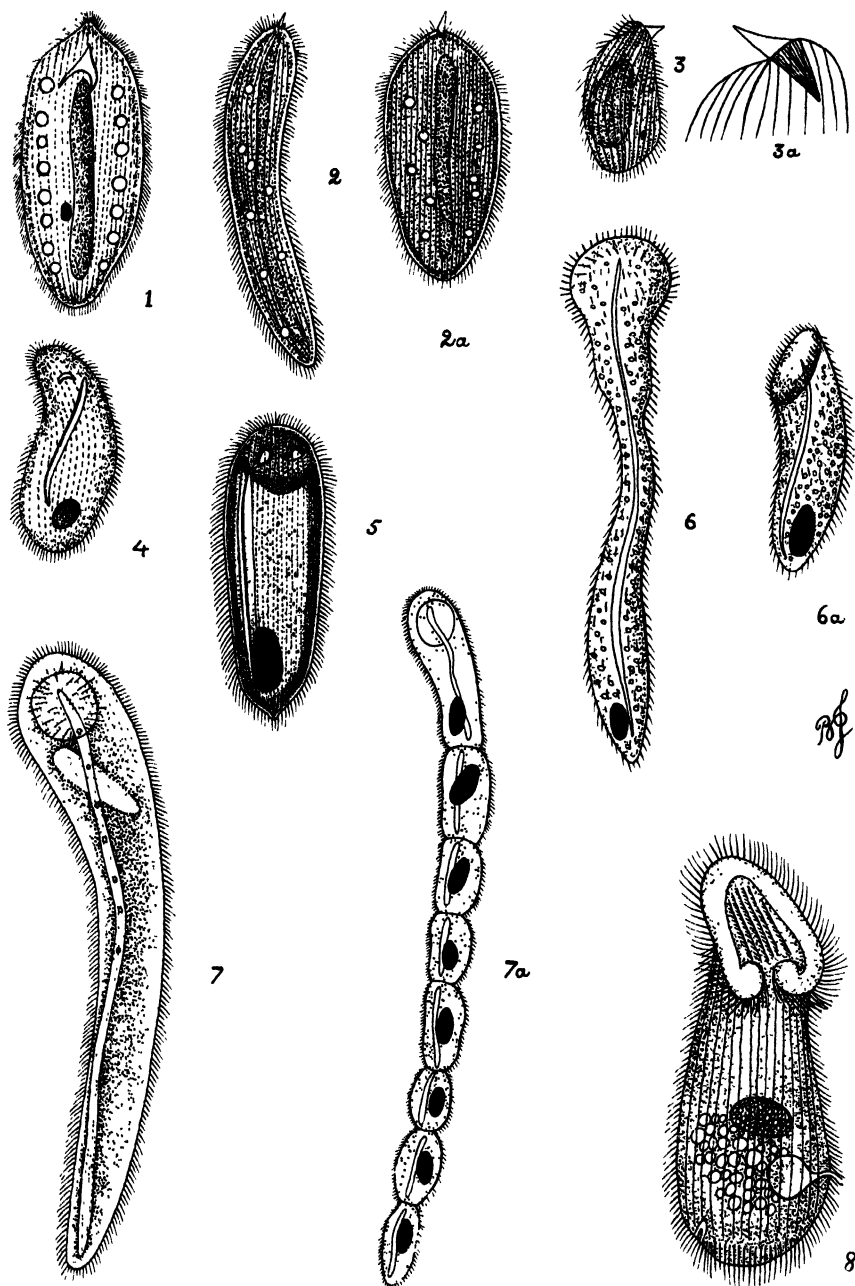
FIG. 493.—VARIOUS ASTOMATEA. (FROM CÉPÈDE, 1910.)

[*For description see opposite page.*

Schultzellina mucronata Cépède, 1910, is one of the smallest astomatous ciliates (Fig. 493, 3). It has an ovoid body which varies in length from 20 to 30 microns. This ciliate, again, is parasitic in the intestine of an earth-worm, *Allurus tetrœdurus.*

4. *Family:* DISCOPHRYIDÆ Cépède, 1910.

The ciliates belonging to this family are characterized by the possession of a sucker, circular in outline, on the ventral surface of the anterior end of the body, and a contractile vacuole in the form of an elongate channel. The sucking disc may or may not be provided with a fixing organ (uncinus), which in some forms is paired, while the disc may or may not be ciliated. Cépède recognizes four genera. The genus *Lachmannella* Cépède includes the single species *L. recurva*, which has actually no sucker, but possesses a single fixation organ or uncinus on the ventral face of the anterior region of the body (Fig. 493, 4). The genus *Steinella* Cépède, including the species *S. uncinata*, has a ventral sucker with two fixation organs, while the two genera *Discophrya* Stein and *Haptophrya* Stein have no uncini, but possess ventral suckers. In the former genus the sucker is bordered by a single row of cilia and in the latter by a double row (Fig. 493, 6-7). The members of the three first-named genera are parasitic in turbellarians, while those of the genus *Haptophrya* occur in newts. *H. gigantea* Maupas, which may reach a length of 1,260 microns, sometimes gives rise to chains of individuals 1,600 microns long (Fig. 493, 7 and 7a).

5. *Family:* LADIDÆ Cépède, 1910.

The single member of this family, *Lada wrzesniowskii*, resembles the members of the preceding family in the possession of a sucking disc, which, however, has a thickened ciliated horseshoe-shaped rim (Fig. 493, 8). It is parasitic in an oligochæte worm.

Parasites of the Liver.

6. *Family:* CEPEDELLIDÆ Cépède, 1910.

This family includes the single genus *Cepedella*, with the one species, *C. hepatica* Poyarkoff, 1909, which occurs in the liver of cyclad molluscs (*Sphœrium corneum*). The body is pear-shaped and pointed at the anterior

1. *Cepedella hepatica* Poyarkoff, 1909 (× 1,609), parasitic in liver of the mollusc, *Sphœrium corneum.*
1a. *C. hepatica,* dividing form (× 2,000).
2. *Herpetophrya astoma* Siedlecki, 1902 (× 500), from body cavity of *Polymnia* sp.
3. *Perezella pelagica* Cépède, 1910 (× 500), from body cavity of marine copepods, showing micro- and macronucleus and single posterior contractile vacuole.
4. *Orchitophrya stellarum* Cépède, 1907 (× 1,300), from the genital glands of the echinoderm *Asteracanthion (Asterias) rubens.*
5. *Protophrya ovicola* Kofoid, 1903 (× 500), from the brood sac and ovaries of the mollusc, *Littorina rudis*—two individuals in a tissue space.
6. *Collinia branchiarum* (Stein, 1852) (× 800), from body cavity of *Gammarus pulex.*
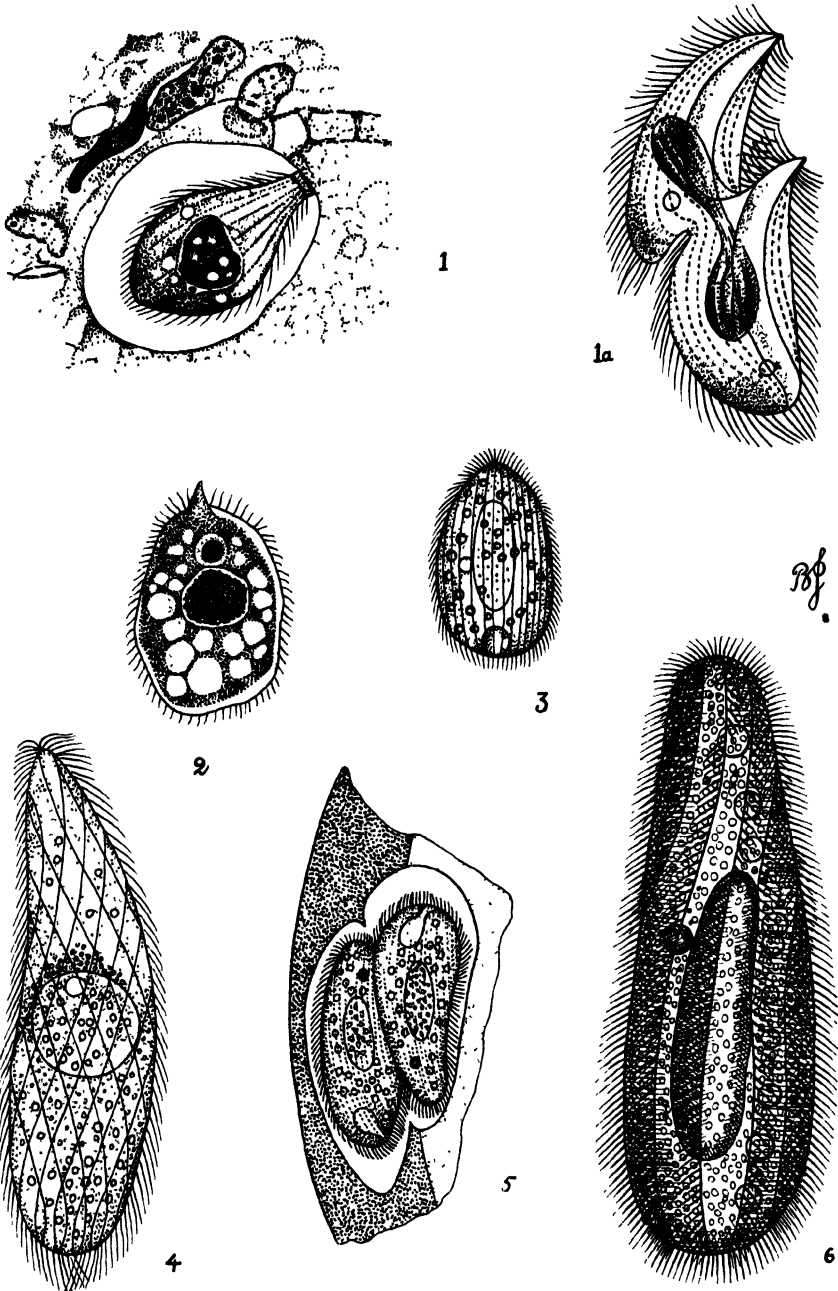
FIG. 494.—VARIOUS ASTOMATEA. (1, AFTER CÉPÈDE AND POYARKOFF, 1909; 2, AFTER SIEDLECKI, 1902, FROM CÉPÈDE, 1910; 3-5, AFTER CÉPÈDE, 1910; 6, AFTER SCHNEIDER, 1886, FROM BÜTSCHLI.)

end, where there is a fixing organ in the form of a concavity from which myonemes radiate (Fig. 494, 1). The ciliate is 16 to 26 microns in length.

Parasites of the Cœlom.

7. *Family:* HERPETOPHRYIDÆ Cépède, 1910.

There is but one genus and one species in this family, *Herpetophrya astoma* Siedlecki, 1902, which is parasitic in the body cavity fluid of an annelid worm of the genus *Polymnia* (Fig. 494, 2). The ciliate has an ovoid body, at the narrow anterior end of which is a pointed, mobile, tactile cone.

8. *Family:* PEREZELLIDÆ Cépède, 1910.

This family, again, contains but a single genus with one species, *Perezella pelagica* Cépède, 1910 (Fig. 494, 3). The ciliates measure up to 76 microns in length. The shape of the body is ovoid except for the fact that the ventral surface is excavated to form a large sucker. The ciliates occur in the body cavity fluid of various copepod crustacea (*Clausia elongata*, *Acartia clausi*, and *Paracalanus parvus*). Cépède has noted what appear to be encysted forms of the ciliate attached to the legs of infected crustacea.

9. *Family:* COLLINIIDÆ, Cépède, 1910.

The members of this family have frequently been ascribed to the genus *Anoplophrya*, from which Cépède has separated them in the genus *Collinia* Cépède, 1910. The members of the genus are parasitic in the body cavity fluids of fresh-water crustacea (*Gammarus*, *Asellus*, *Neoniphargus*). They have ovoid bodies devoid of cytostome, a small number of longitudinally arranged rows of cilia. and contractile vacuoles which vary in number according to the size of the body.

Collinia branchiarum (Stein, 1852) is parasitic in *Gammarus pulex;* *C. neoniphargi* Cépède, 1910, in *Neoniphargus moniezi;* and *C. circulans* (Balbiani, 1885), in *Asellus aquaticus*. The largest form is *C. branchiarum*, which reaches a length of 120 microns, while *C. circulans* varies in length from 16 to 50 microns. *C. neoniphargi* reaches a length of 60 microns.

The first form noted was seen by Stein (1852) in the body cavity of *Gammarus pulex*, and was named by him *Opalina branchiarum* (Fig. 494, 6). Balbiani (1885) discovered a similar parasite in *Asellus aquaticus*, and named it *Anoplophyra circulans*. A third was recorded from *Neoniphargus moniezi* by Cépède (1910), who placed it in the genus *Collinia* as *C. neoniphargi*. He also placed in this genus the two species previously described. The differences between the three species are merely those of size, and as Brumpt (1913*d*) points out, it must be doubtful if this is
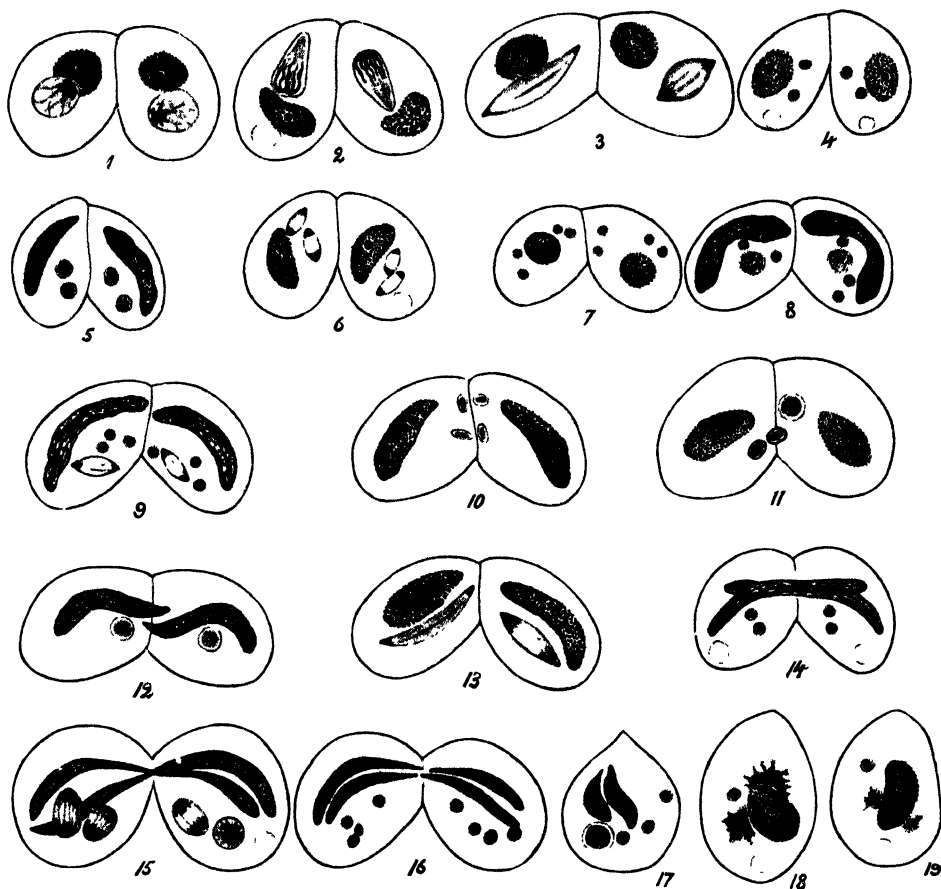
FIG. 495.—*Collinia branchiarum :* VARIOUS STAGES IN CONJUGATION (× *ca.* 2,000).
(AFTER COLLIN, 1909.)

1-7. Changes in macronucleus and multiplication of micronucleus till four are present in each ciliate.
8-9. One of the daughter micronuclei in each ciliate increases in size and divides, while the others degenerate.
10. Three of the daughter micronuclei have degenerated, while one divides to form the conjugating nuclei.
11-12. The migratory nucleus passes over and unites with the stationary nucleus.
13-16. The single nucleus divides, and eventually four are produced in each conjugant. The macronuclei, much elongated, lie parallel to one another across the point of union of the conjugants. At this stage the conjugants separate, each receiving a half of each macronucleus.
17. One of the separated conjugants. The macronuclei degenerate, while one of the micronuclei enlarge to form a new macronucleus.
18. The ciliate divides after division of the macronucleus. The micronuclei do not divide, so that one daughter individual has two micronuclei and the other only one. The one with two micronuclei presumably divides again in a similar manner, producing two ciliates, each with one macronucleus and one micronucleus. The ciliates then multiply in the usual manner after division of both nuclei.

a sufficient basis for separating them. The macronucleus is an elongate structure, and near it is a micronucleus. Several contractile vacuoles are present. Cépède (1910) gave the measurements of *C. branchiarum* as reaching 120 microns, *C. neoniphargi* as 60 microns, and *C. circulans* as 16 to 50 microns. Reproduction by equal binary fission or by a budding process leading to chains of buds occurs, while conjugation has been studied by Collin (1909) in *C. branchiarum*, and by Schneider (1885) and by Brumpt (1913*d*) in *C. circulans*.

According to Brumpt, active multiplication by binary fission precedes the conjugation process in *C. circulans*, so that small individuals measuring about 10 by 7 microns result. During conjugation of *C. circulans* and *C. branchiarum* two ciliates become associated, and their micronuclei divide twice to form four nuclei, while the macronuclei elongate (Fig. 495). Three of the small nuclei in each individual degenerate, while the remaining one divides again. Each individual has now two small nuclei and an elongate macronucleus. One of the small nuclei of each conjugant then passes into the other and unites with its stationary nucleus. At the same time the elongate macronuclei become arranged side by side, so that they extend across the point of union of the conjugants. The point of union then becomes narrower and the ciliates separate. In so doing they cause the macronuclei to divide, so that each ciliate acquires half of each macronucleus. The two half macronuclei in each ciliate now become roughly spherical and gradually disintegrate. The synkarion formed by the union of the two micronuclei now divides, and the two nuclei so formed divide again to give four micronuclei. Of these four, two degenerate and disappear. One becomes transformed into the new macronucleus, while the other remains as the micronucleus. It is probable that at the nuclear division which immediately precedes the conjugation of the micronuclei the number of chromosomes is reduced, but Brumpt makes no mention of this in *C. circulans*.

In the case of *C. branchiarum*, Collin (1909), who was the first to describe accurately the process of conjugation, noted that the number of chromosomes in the micronuclei of ordinary dividing individuals was six. In conjugation at the first division of the micronucleus each of the six chromosomes divides, so that each daughter nucleus has six. At the second division the chromosomes do not divide, but three pass to each daughter nucleus, so that the number is halved. This is the true reducing division. At the next division each of the three divides, so that the daughter nuclei, which are the conjugating nuclei, again have three chromosomes. When the nuclei unite, the resulting synkarion has six chromosomes, and this number is maintained at all subsequent divisions till the next conjugation occurs. In view of the fact that they ultimately

degenerate, it is difficult to account for the behaviour of the macronuclei during conjugation as described by Schneider, Collin, and Brumpt.

Under certain circumstances the ciliates become encysted, and it is evident that these stages are responsible for the spread of infection

10. *Family :* PROTOPHRYIDÆ Cépède, 1910.

This family includes the forms *Protophrya ovicola* Kofoid, 1892, and *Isselina intermedia* Cépède, 1910, which are parasitic in molluscs of the genus *Littorina* (Fig. 494, 5). The former occurs in the uterus and the latter in the mantle cavity. In these forms food is apparently ingested through an opening (cytostome) at the posterior end of the body.

11. *Family :* ORCHITOPHRYIDÆ Cépède, 1910.

This family was established by Cépède to include the genus *Orchitophrya*, which contains the single species *O. stellarum*, parasitic in the reproductive glands of the star-fish, *Asteracanthion (Asterias) rubens* (Fig. 494, 4).

(2). *Sub-Order :* **Stomatea**.

In this sub-order are included ciliates which have the body either completely, or only partly, covered with cilia, which vary little in length and thickness in different regions. Furthermore, a cytostome is present, and this is either devoid of cilia and capable of being closed during the intervals between feeding (*Section* 1 *:* GYMNOSTOMATA, Bütschli, 1889), or it is permanently open and has within it cilia, which may be fused to form a membrane (*Section* 2 *:* TRICHOSTOMATA, Bütschli, 1889). Amongst the Gymnostomata are found free-living ciliates belonging to the genera *Chilodon, Holophrya, Nassula, Didinium, Prorodon, Coleps, Lacrymaria,* and others, and certain parasitic forms, such as *Bütschlia, Ichthyophthirius,* and possibly others, such as *Blepharosphæra* and *Blepharocodon* and their allies. Amongst the Trichostomata there are numerous free-living ciliates belonging to the well-known genera as *Paramecium, Pleuronema, Glaucoma, Colpoda, Colpidium,* and parasitic forms, such as *Isotricha, Dasytricha,* and others. Many of these parasitic forms from the stomach of cattle or the cæcum of horses require further investigation from the point of view of the arrangement of the cilia in and around the cytostome.

Genus : **Chilodon** Ehrenberg, 1833.

The ciliates belonging to this genus are of interest, for the skin of fish may be heavily infested with them, and though they do not penetrate the skin, they may occur in such numbers on the gills that respiration is impaired. Certain forms have been seen in dysenteric or diarrhœic

stools of human beings, but there is no evidence that the ciliates actually occurred in the human intestine. It is possible that they may pass through the intestine in the encysted condition, and give rise to cultures in the fæces.

A typical member of the genus, such as *Chilodon cucullulus*, has a dorsal and ventral surface, the former being convex and the latter flat (Fig. 496). Observed from above, the outline of the body is not symmetrical, for one side is straight or slightly concave, while the other is convex. It is
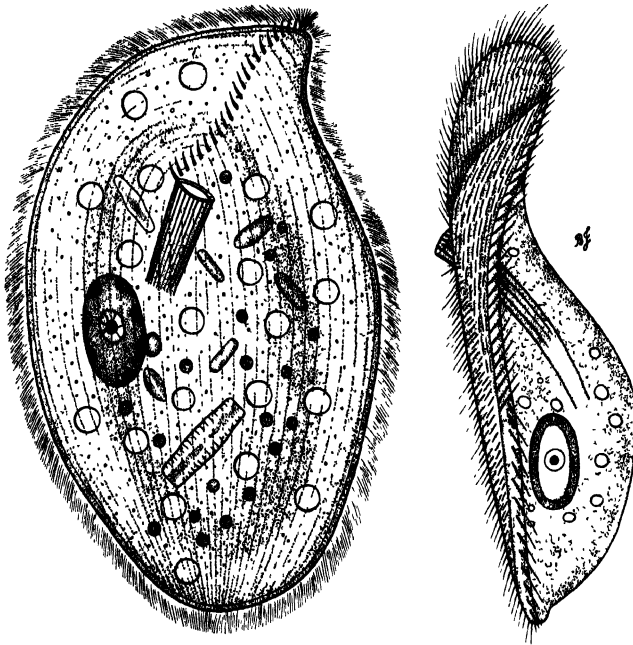
Fig. 496.—*Chilodon cullulus*: Ventral and Side View (× *ca.* 3,000).
(After Schewiakoff, 1896, and Stein, 1859.)

almost kidney-shaped in outline, but the anterior end is broader than the posterior. The anterior margin forms a thin lip, which passes along the convex left margin of the body and loses itself near the posterior end. The whole of the ventral surface is covered with cilia, as also the margins.of the dorsal surface. Towards the anterior end of the ventral surface is the cytostome leading to a straight or slightly curved œsophagus, which is supported by longitudinal bars representing thickenings of the cytoplasm. They are known as trichites, while the part of the œsophagus where they occur is called the oral basket. There is an ovoid macronucleus and a small micronucleus. The ciliates produce spherical cysts.

Ehrenberg (1833) recognized a form, *Chilodon uncinatus*, and placed in this genus as *C. cucullulus* the ciliate named *Kolpoda cucullulus* by O. F. Müller (1773). Fromentel (1874) named a form seen by him *C. dentatus*, but it seems probable this was actually *C. uncinatus*, in which the supporting apparatus of the œsophagus is continued into the cytoplasm, where it pursues a spiral course (Fig. 497). The division and conjugation of *C. uncinatus* has been studied by Enriques (1908a) and MacDougall (1925). MacDougall, as did Kent and Enriques, noted that when transverse division takes place the old oral basket disintegrates, a new one being formed for each of the daughter ciliates. When conjugation occurs, the two ciliates unite by the oral apertures, but the oral baskets degenerate, a new one being formed in each ciliate before they separate. As regards the nuclear changes during syngamy, the micronucleus undergoes maturation divisions. In the first of these, as noted by Enriques (1908), there are four chromosomes, all of which divide. The survivor of the two daughter nuclei then divides, but on this occasion the four chromosomes do not divide, two of them passing to each daughter nucleus. One of these nuclei degenerates, and the other one divides again. The two chromosomes both divide, so that the two daughter nuclei, which are those which take part in the syngamic process, each have two chromosomes. After the migrating nuclei have united with the stationary nuclei the two nuclei which result each have four chromosomes, this number, which is the

FIG. 497.—*Chilodon uncinatus* AS SEEN WHEN FIXED AND STAINED (× *ca.* 2,500). (AFTER MACDOUGALL, 1925.)

The œsophagus is supported by rods (trichites), the whole forming the oral basket. Posteriorly the trichites merge into the wall of the œsophagus, which takes a spiral course in the cytoplasm. The cilia are not shown, though their point of insertion are indicated by dots.

diploid number, being retained through all the subsequent divisions of the ciliates. MacDougall noted that in a certain large race of *C. uncinatus* the diploid number of the chromosomes was eight, and that the conjugating nuclei each had four. The individuals of this race resembled those of the race with half the number of chromosomes in every detail except size. As this form appeared in a culture of the smaller type, it appeared probable that it had resulted from a conjugation in which for some reason the reduction in the number of chromosomes had not taken place.

Guiart (1903) identified as *C. dentatus* (*C. uncinatus*) a ciliate which he saw in a dysenteric stool in France. Suspecting that the ciliates might have had an extraneous origin, a fresh specimen was obtained, and in this no ciliates were found. Some of the material, however, placed in sterile water produced a rich culture of the ciliate, an observation which suggests the presence of encysted forms in the fæces.

Manson and Sambon (1909) recorded the presence of *C. uncinatus* in the stool of a case of schistosomiasis. There is no evidence that it was present in the freshly-passed stool.

Seleneff (1910) ascribed to the genus *Chilodon* a ciliate which he claimed to have found in the prostatic secretion of four patients who were suffering from urethritis. The ciliates were said to be present in the secretion after the urethra had been irrigated and the prostate compressed. It seems not improbable that the irrigating fluid may have been contaminated with a common free-living ciliate.

As regards the forms which infest the skin of fish, these may be the well-known species. On the other hand, two of them have been given specific status—*C. cyprini* Moroff, 1902, and *C. hexastichus* Kiernik, 1909. According to the descriptions. they differ from *C. uncinatus* in minor details only.

Genus : **Colpoda** O. F. Müller, 1773.

The ciliates belonging to this genus, several species of which are known, are very common organisms in stagnant water and moist earth, and it is not surprising that fæcal material received into vessels which are not sterile may become contaminated. This is all the more likely to occur, as the various species of *Colpoda* readily encyst; in fact, the ordinary reproduction by fission takes place in the encysted condition, as first shown by Stein (1854). The ciliates are bean-shaped and flattened dorso-ventrally (Fig. 498). The cytostome lies at the end of a groove on the right-hand side of the ventral surface in front of the middle of the body. The posterior margin of the groove is armed with a row of long cilia, which are flexible only at their distal ends. The flexible ends protrude beyond the margin of the body, and form a backwardly directed tuft or brush. The anterior margin of the groove is furnished with short cilia. Cilia are also present in the cytopharynx and round the margin of the cytostome. The side of the body in front of the cytostomal groove is notched or lobed, each notch corresponding with one of the longitudinal rows of cilia which cover the body. The part of the body in front of the cytostome is much thinner dorso-ventrally than the part behind it, which is more globular in shape. There is a contractile vacuole at the posterior end. The macronucleus, situated in the posterior thicker

half of the ciliate, is an elongate or spherical structure possessing a central deeply-staining spherical body resembling a karyosome. Sometimes several smaller bodies of a similar nature are present in addition. Near the macronucleus is the small micronucleus. A form seen by Schulz (1899) in human fæces in Germany was identified by him as *C. cucullus* Ehrenberg. Yakimoff and Kolpakoff (1921) identify as *C. steini* Maupas a form in human fæces in Russia. There is no reason to suppose that either of these is parasitic.
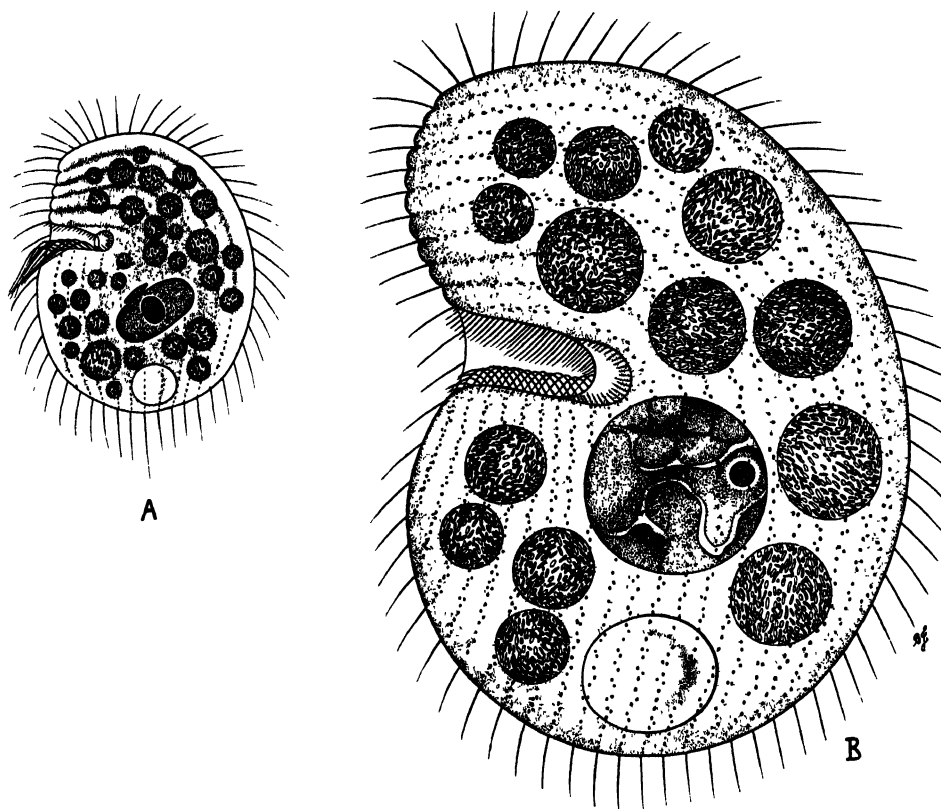
FIG. 498.—*Colpoda steini* (A) AND *Colpoda cucullus* (B): VENTRAL VIEWS (× 1,500)
(ORIGINAL.)

Species of *Colpoda* rapidly appear in water in which hay is placed. They grow readily in this infusion, as also on the surface of agar plates, where they live by ingesting bacteria. Unlike the majority of ciliates, they multiply only in the encysted condition. *C. steini*, which has been studied by the writer, when about to divide, becomes roughly spherical in form, and, revolving continuously, secretes around itself and beyond its cilia a clear transparent cyst which measures from 12 to 32 microns in

diameter (Figs. 38 and 499). The macronucleus and the micronucleus divide, after which the ciliate itself is split into two. A new contractile vacuole appears in one half. Division of the nuclei again takes place in each of the daughter forms, which then divide. New contractile vacuoles appear in those which have not acquired the ones present in the parent form. During the whole of this process the contents of the cyst are revolving by the action of the cilia which are not lost at any stage. After the four ciliates are formed, some time is occupied in their complete re-organization, after which their movement becomes increasingly violent till the cyst is ruptured and they escape. The whole process occupies about six or seven hours. Cysts of a purely protective nature are also produced. These have thick walls, and can withstand prolonged desiccation. The writer has seen two ciliates become attached to one another by their cytostomes and form a common cyst. Though the subsequent changes were not followed, it seems probable that this represented the commence-ment of syngamy.

According to Enriques (1908), there are three species of *Colpoda*, which can be distinguished from one another by the number of lobes in the pre-cytostomal region, the character of the macronucleus and its karyosome, and the size of the body.

C. cucullus is 40 to 100 microns in length, and has nine to ten lobes in the frontal region. The macronucleus is spherical, and possesses a lobed " karyosome " (Fig. 498, B).

C. maupasi is 35 to 70 microns in length, and has six to seven lobes in the frontal region. The macronucleus resembles that of *C. cucullus*.

C. steini is 23 to 48 microns in length, and has six to seven lobes in the frontal region. The macronucleus is elongate, and has a spherical or ovoid " karyosome " (Fig. 498, A).

Enriques observed conjugation in *C. steini*, but not in the other forms.

Genus: Uronema Dujardin, 1841.

The ciliates belonging to this genus are free-living organisms which have ovoid bodies uniformly covered with cilia and, laterally placed on the right-hand side of the ventral surface, a buccal cavity provided with a membrane. There is a posterior flagellum-like caudal process. They have occasionally been described from human fæces, and a ciliate which may belong to this genus has been found in the body cavity of small aquatic Crustacea.

Martini (1910) gave the name *Uronema caudatum* to a ciliate seen by him in the stools of a dysenteric case in China. More recently Yakimoff (1921) claims to have seen four examples of the infection in Russia. There can be no doubt that in these cases the stools had become
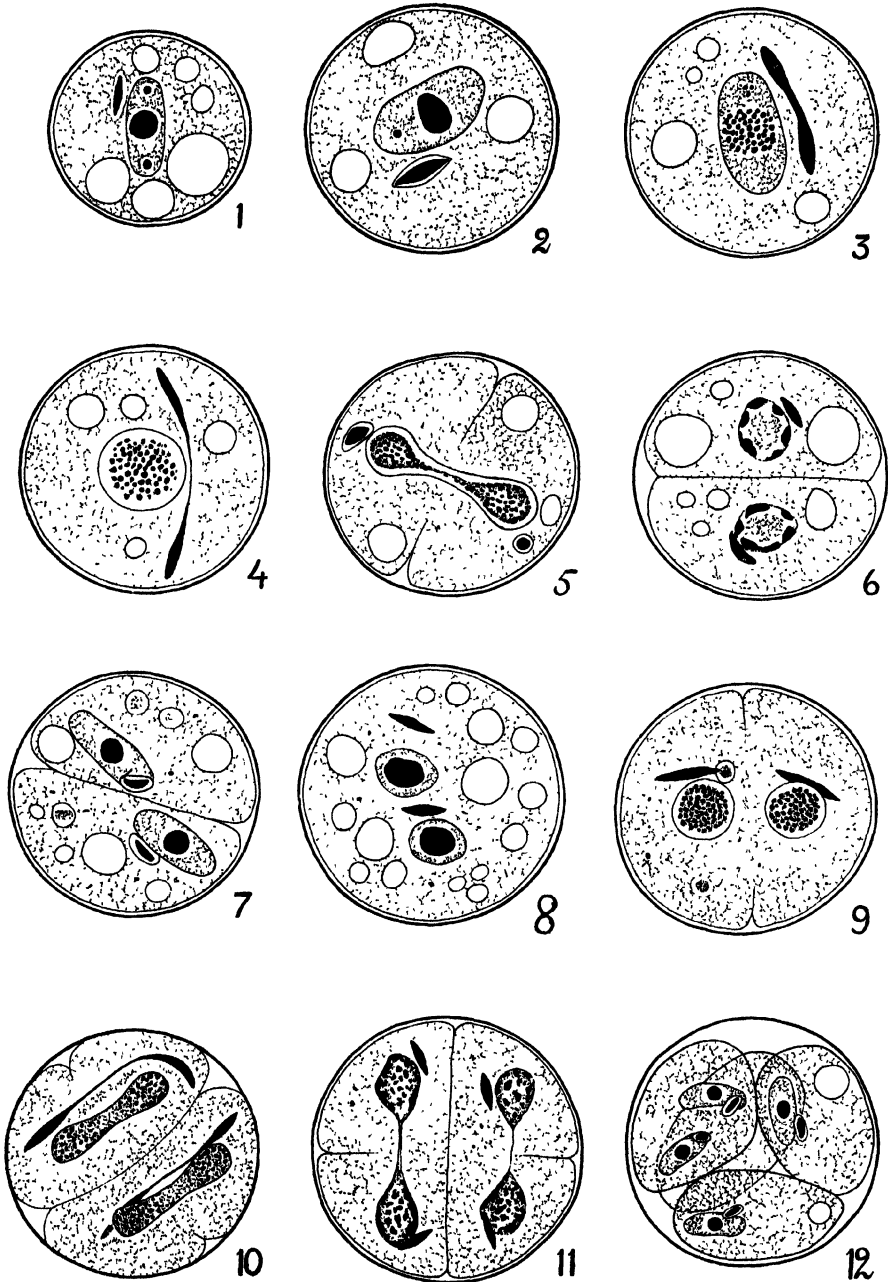
FIG. 499.—*Colpoda steini* (× 1,600): DIVISION IN CYST TO PRODUCE FOUR DAUGHTER INDIVIDUALS. THE CILIA WHICH PERSIST THROUGHOUT ARE NOT DEPICTED. (ORIGINAL.)

contaminated with a free-living ciliate, or its cysts, for Yakimoff only obtained the organism by inoculating culture media with human fæces. In fact, Yakimoff's ciliate does not appear to belong to the genus *Uronema*, while Martini's is probably a *Cyclidium,* as noted by Dobell (see Dobell and O'Connor, 1921).

The type species *U. marina* is ovoid in form, slightly flattened dorso-ventrally, and measures 40 to 50 microns in length (Fig. 500 B). The anterior end is more pointed than the other. The body is uniformly covered
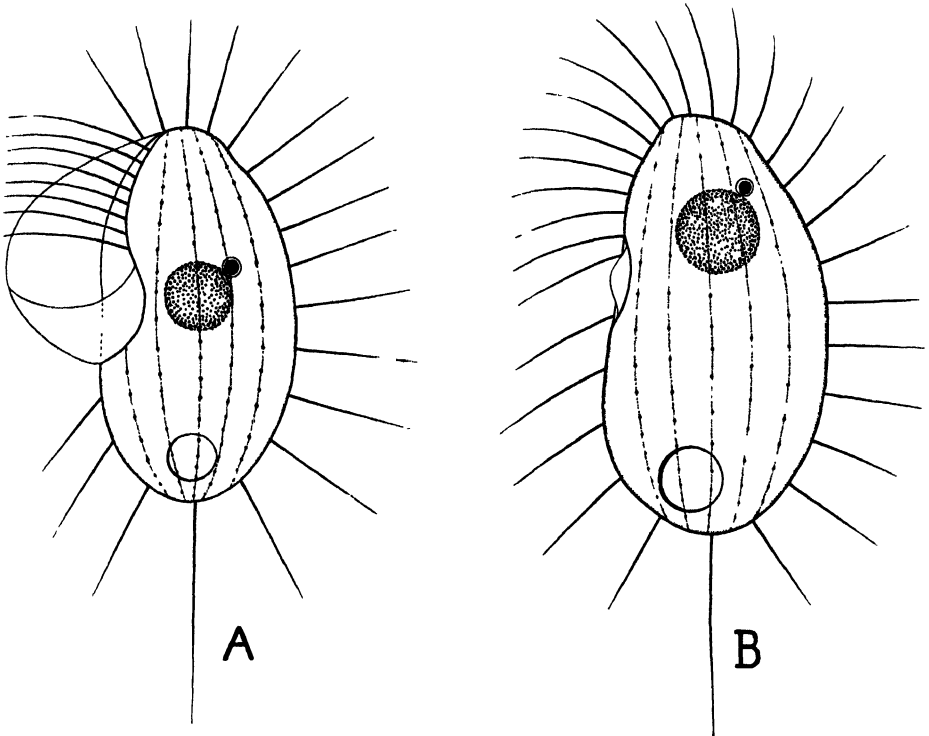
FIG. 500.—CILIATES OF THE GENERA *Cyclidium* AND *Uronema* (× *ca.* 1,500).
(ORIGINAL.)

A. *Cyclidium glaucoma.*   B. *Uronema marina.*

with longitudinal rows of cilia, while posteriorly there is a caudal process which may be as long as the body itself. The cytostome is at the right-hand side of the ventral surface. Its opening is surrounded with cilia, which are longer than those on the rest of the body, while in association with it is a membrane which, by its undulating movements, directs food into the cavity. There is a spherical macronucleus, beside which is the micronucleus. A contractile vacuole is present at the posterior end of the body.

Cépède (1910) observed that marine copepod Crustacea were liable to be invaded by a ciliate which he named *Uronema rabaudi*. Not only did the ciliate occur in the dead crustacea, but also in the living ones.

Genus : **Cyclidium** O. F. Müller, 1773.

The ciliates belonging to this genus resemble species of *Uronema* in many respects (Fig. 500, A). They are about the same size, are similarly ciliated, and possess a caudal process. There is, however, a large peristome, while the membrane is attached to the whole length of the right margin, as also to the posterior part of the left margin, forming a kind of pocket. A ciliate which was discovered by Mac Arthur (1922) in larvæ of *Theobaldia annulata* was ascribed to this genus, but it seems probable that the ciliates actually in the larvæ belonged to the genus *Glaucoma*, and that a species of *Cyclidium* was present in the water.

The ciliates of the genus *Pleuronema* are very similar. They are larger, however, than *Cyclidium*, have a more extensive membrane, and are devoid of the caudal process.

Genus : **Glaucoma** Ehrenberg, 1830.

The members of this genus are ovoid in form, and from 20 to 80 microns in length. They are uniformly ciliated, and have a relatively small funnel-shaped peristome provided with a membrane which, in some forms at least, has its anterior border strengthened by a finger-like process. At the bottom of the peristome is the cytostome. Ciliates of this genus are common in infusions and stagnant waters, and they grow readily on agar plates.

It seems probable that the majority of the ciliates which invade the body cavities of insect larvæ, and which have been described under various names, belong to this genus.

Mac Arthur (1922) discovered ciliates in the body cavity of larvæ of *Theobaldia annulata* in England. They were found in both living and dead larvæ, the head region of the body containing large numbers of ciliates, which extended into the antennæ and other appendages (Fig. 501). They appeared to have a special predilection for attacking the eyes, which became disorganized completely by their activities. Though the ciliates which invade the larvæ multiply very rapidly in the body cavity, they are not entirely parasitic, as they will live and multiply indefinitely in water or any suitable medium. It is possible that the ciliates were eaten by the larvæ, and that in certain sick individuals they had penetrated the gut wall and entered the body cavity, where, by their rapid multiplication, they had hastened the death of the larvæ. The writer was able to study this ciliate, which was

kindly supplied to him by its discoverer. He was successful in keeping cultures in water, hay infusions, and in the liquid on the surface of agar plates. It seems probable that in the cultures a mixture of two organisms occurred, one of them being a species of *Cyclidium* and the other a species of *Glaucoma*, probably *G. pyriformis*. The forms actually present in the

FIG. 501.—HEAD OF *Theobaldia annulata*, SHOWING INVASION BY *Glaucoma pyriformis* (× 100). (AFTER MAC ARTHUR, 1922.)

larvæ (Fig. 502, A) had the usual appearance of *G. pyriformis* after fixation. There was a small buccal cavity in which a minute compact body could be detected. It probably represented the shrunken membrane and finger-like process which can be seen in the living organism, or possibly a second membrane, which is described by some observers.

During life the finger-like process, which is attached to the anterior wall of the buccal cavity near its opening, pivots about its attachment in such a manner that it is sometimes within the buccal cavity and at other times outside it and pointing in a direction at right angles to the margin of the body. There is a triangular membrane, the attachments of which are variously described. It is adherent to the whole length of the finger-like process, which may be regarded as a thickening of this margin of the membrane. Another margin of the membrane is fixed either to

A B
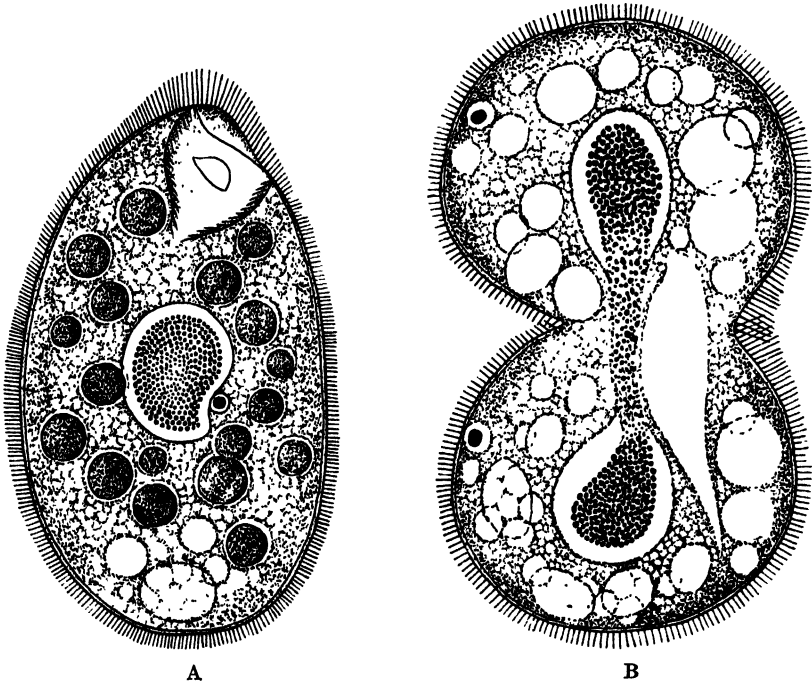
FIG. 502.—*Glaucoma pyriformis* FROM THE HEAD OF THE LARVÆ OF *Theobaldia annulata* (× 2,000). (AFTER MAC ARTHUR, 1922.)

A. Usual type. B. Dividing form.

the right lip of the buccal orifice, or, as Mac Arthur figures it, to its anterior wall. The other margin of the membrane is free. When the finger-like process is pointing outwards the membrane is extended and clearly visible, but when it is turned back into the buccal cavity the membrane is taken with it. A second and smaller membrane is also described as being within the buccal cavity, and capable of being extended through its orifice as a tongue-like process.

The organism reproduces in the usual manner by binary fission

(Fig. 502, B). After division of the micro- and macronucleus, a constriction appears at the middle of the body, and two ciliates result. A new peristome is formed for the posterior individual, and a new contractile vacuole for the anterior one. Recently Treillard and Lwoff (1924) have found a similar infection of larvæ of *Chironomus plumosus*. The ciliate in this case corresponded with *Glaucoma pyriformis*, so that it is possible that the form in the larvæ of *Theobaldia* was this species. Lwoff (1924) has shown that if the caterpillar of *Galleria mellonella* is inoculated with a culture of *Glaucoma pyriformis*, the ciliate reproduces actively and brings about a fatal infection.

A ciliate which was parasitic in the body cavity of the larvæ of *Aëdes* (*Stegomyia*) *scutellaris* in the Malay States was discovered by Lamborn (1921). Keilin (1921a) examined fixed material which was sent him, and proposed the name *Lambornella stegomyiæ* for the ciliate. The infected larvæ were noted by Lamborn to be paler than healthy ones, while none of them reached maturity. The ciliates occurred in all parts of the body cavity, and extended especially into the siphon and gills. According to Keilin, they varied in length from 50 to 70 microns, with a breadth of 30 to 40 microns. There was a rounded macronucleus and a micronucleus. A small structure seen at one side near the anterior end of the body was taken for the cytostome. It seems to the writer that this structure is actually something within the peristome, for an exactly similar body was seen in the same position in ciliates within fixed *Theobaldia* larvæ infected with the organism described above. It is possible that this structure is actually the retracted finger-like process. On the outside of the cuticle of one larva Keilin observed certain flattened cysts 30 to 40 microns in diameter, which he concluded were cysts of the ciliate. Keilin, who only examined imperfectly fixed material, was unable to make out the details of structure which are essential for the identification of a ciliate, so that his genus *Lambornella* is actually without definition. It is quite possible that the ciliate is the same as the one discovered by Mac Arthur in the larvæ of *Theobaldia*, and Treillard and Lwoff in larvæ of *Chironomus plumosus*, but till more accurate details are forthcoming it will be impossible to settle this point.

Another parasitic ciliate is *Glaucoma parasiticum*, which Penard (1922) discovered in the gills of the aquatic crustacean, *Gammarus pulex*. It varied in length from 35 to 70 microns, and it appears to resemble closely *Glaucoma pyriformis*.

Lichtenstein (1921) described as *Ophryoglena collini* a much larger ciliate, which was parasitic in ephemerid larvæ (*Bætis*). It was ovoid in form, and measured 200 to 300 by 120 to 230 microns. It destroyed the

tissues of the body, especially those of the generative organs. The cyto-stome (? peristome) is a small horseshoe-shaped aperture towards the anterior end of the body. There is an undulating membrane in the cytopharynx, and a refractile structure shaped like a watch-glass on the left side of the cytostome. There was a large sausage-shaped macro-nucleus and a small micronucleus.

Genus: Ichthyophthirius Fouquet, 1876.

The ciliates belonging to this genus are parasitic in the skin of fish. There is an anterior cytostome, the ovoid body is covered by longitudinal rows of cilia, and multiplication takes place within cysts by repeated binary fission.

Ichthyophthirius multifiliis Fouquet, 1876.—This is a ciliate which produces a cutaneous pustular eruption on the skin of various fresh-water fish (Fig. 503). The ciliate fixes itself to the skin and gradually becomes embedded in the epidermis. As it increases in size, a tiny white pustule is produced. Eventually this ruptures, and the enclosed ciliate and other contents are discharged into the water. When the fish are heavily in-fected, as is sometimes the case, the whole body is covered with the pustules, and when the gills are involved, the health of the fish is seriously impaired.

The ciliate is egg-shaped, and measures from 500 to 800 microns in length (Fig. 503, 1). There is a small cytostome leading to a short œsophagus at the anterior and more pointed end, and a cytopyge at the posterior end. The body is uniformly covered with longitudinal rows of cilia, and there are numerous superficial contractile vacuoles. There is a large horseshoe-shaped nucleus, but a micronucleus has not been seen in the fully-grown forms.

There is some doubt as to whether multiplication of the ciliate can take place within the pustules, for the presence of two parasites in a single pustule, as sometimes occurs, may be merely an indication of an invasion of one spot on the skin by two ciliates (Fig. 503, 5). When the pustule ruptures, the fully-grown ciliate escapes into the water, sinks to the bottom, and becomes enclosed in a thick gelatinous sheath or cyst, within which multiplication by repeated division takes place. In this manner is produced a very large number (100 to 1,000) of small ciliates 30 to 50 microns in length (Fig. 503, 2-4). Each possesses a single contractile vacuole, an ovoid macronucleus, and a small spherical micronucleus. The micronucleus, which is not evident in the fully-grown form, is described as arising from the macronucleus in the course of the nuclear divisions within the cyst. It is supposed by Bütschli (1882-1889), Neresheimer (1908), and Prowazek (1920) that the micronucleus divides

into two, and that after each of the daughter nuclei has undergone a reduction of chromatin the two nuclei reunite (autogamy). Finally, the micronucleus fuses with the macronucleus. It is extremely doubtful if such a process actually occurs. These small ciliates invade the skin of
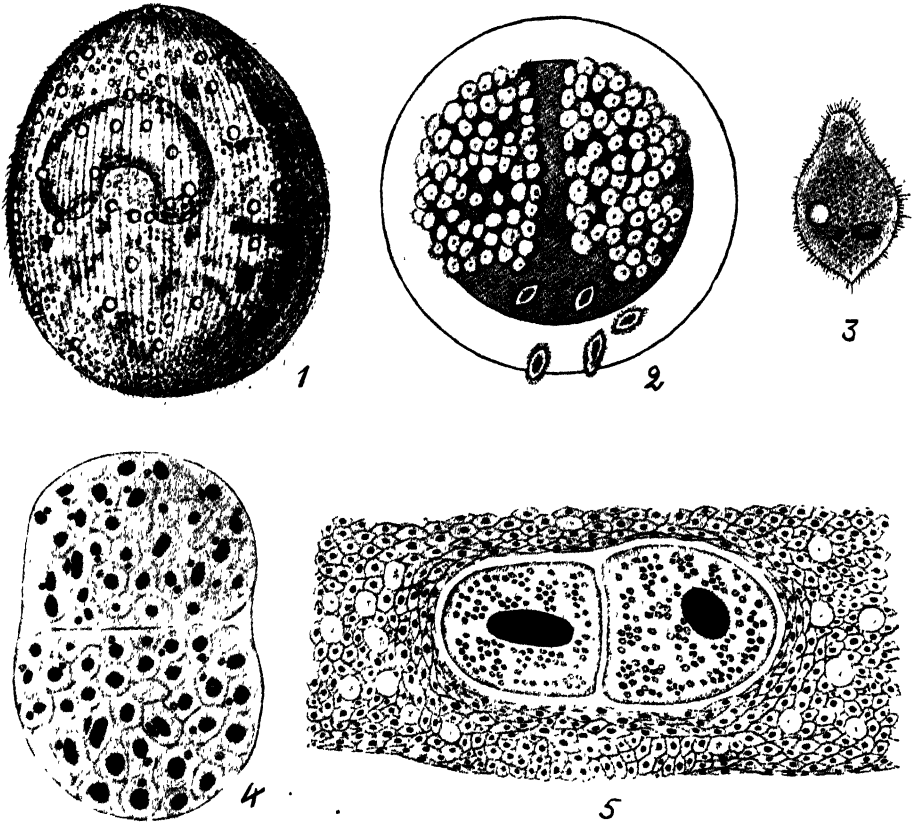
FIG. 503.—*Ichthyophthirius multifiliis* FROM THE SKIN OF FISH. (1-3, AFTER BÜTSCHLI, 1887-1889; 4-5, AFTER DOFLEIN, 1901.)

1. Adult ciliate (× 75).   2. Mature cyst filled with daughter ciliates (× 75).
3. One of daughter ciliates from cyst more highly magnified.   4. Section through a cyst.
5. Section of skin of carp, showing two ciliates in a vacuole.

the fish, which they penetrate by performing a continuous rotary movement at one spot. Embedded in the skin, they grow into the adult form within the pustules.

Genus: **Bütschlia** Schuberg, 1888.

The ciliates belonging to this genus are parasitic in the rumen of cattle, where they were first noted by Schuberg (1888), or the cæcum of the

horse, as described by Bundle (1895). A typical organism is egg-shaped, and there is a terminal cytostome leading to a short œsophagus. A large spherical nucleus is present, and also a contractile vacuole. Schuberg, in the case of *B. parva*, could detect cilia only at the anterior end of the body, but later observers believe that the whole body is covered with them. Three species have been described from the stomach of cattle and one from the cæcum of the horse (Fig. 504, 1-2).

Genus : Holophryoides Gassowsky, 1919.

This genus contains the single species, *H. ovalis*, which occurs in the cæcum and colon of the horse. Fiorentini (1890), who first saw it, placed it in the genus *Paraisotricha*. Gassowsky (1919) established the new genus for its reception. It appears to be closely related to members of the preceding genus. The ciliate, which measures 95 to 140 microns in length by 65 to 90 microns in breadth, has an ovoid or ellipsoidal body, which is covered with cilia (Fig. 504, 3). There is a cytostome at the conical anterior end, and a cytopyge at the posterior end, near which lies the single contractile vacuole. The macronucleus and micronucleus are at the centre of the body.

Genus : Blepharozoum Gassowsky, 1919.

There is a single species, *B. zonatum*, which was seen by Gassowsky (1919) in the cæcum of the horse (Fig. 504, 4). The body, which measures 230 to 245 by 115 to 122 microns, is ovoid and covered with fine cilia. When the ciliate swims the cilia are uniformly active, except along two bands or zones which encircle the body, so that there are three regions in which the cilia are in movement. The cytostome is near the blunter end of the body, and there is a posterior cytopyge. There are two to four contractile vacuoles.

Genus : Prorodonopsis Gassowsky, 1919.

The single member of this genus, *P. coli*, inhabits the colon of the horse (Fig. 504, 5). It is ovoid in shape and covered with cilia. The cytostome is at the anterior, more pointed end, and there are two or three contractile vacuoles. The ciliate measures 55 to 67 by 38 to 45 microns.

Genus : Paraisotrichopsis Gassowsky, 1919.

This genus, of which there is a single species, *P. composita*, is ovoid in shape and uniformly ciliated (Fig. 504, 6). There is an anterior cytostome, near which commences a groove which takes a somewhat spiral course to the posterior end of the body. The ciliate, which measures 43 to 56 by 31 to 40 microns, occurs in the cæcum of the horse.
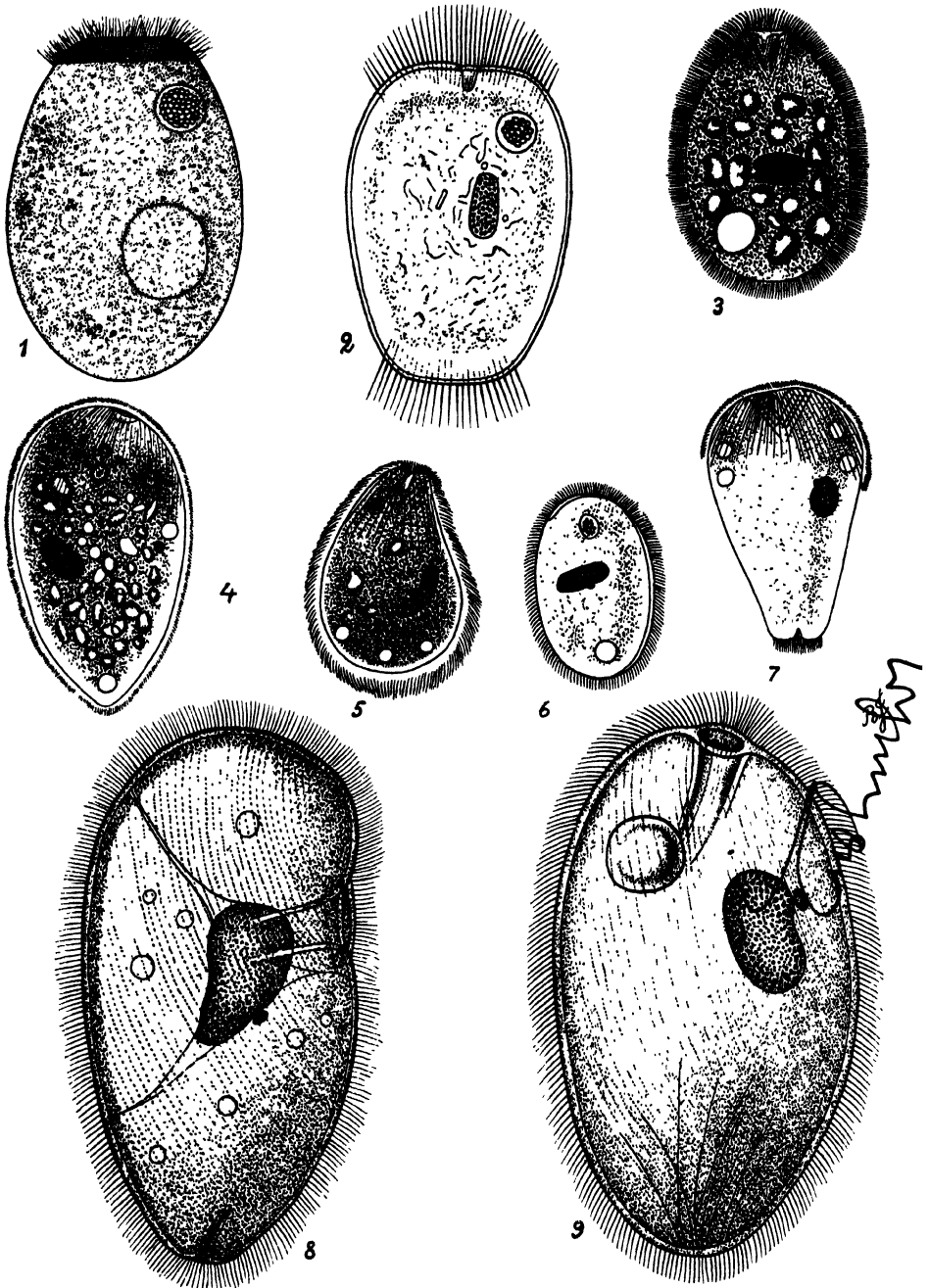
FIG. 504.—HOLOTRICHOUS CILIATES FROM THE INTESTINE OF CATTLE AND HORSES.
(1 AND 9, AFTER SCHUBERG, 1888; 8, AFTER EBERLEIN, 1895; 2, AFTER BUNDLE,
1895; 3-7, AFTER GASSOWSKY, 1919.)

Genus: **Blepharoconus** Gassowsky, 1919.

The single representative of this genus, *B. hemiciliatus*, occurs in the colon of the horse (Fig. 504, 7). It is somewhat conical in shape, and measures 83 to 135 by 45 to 65 microns. The cilia are limited to the anterior part of the body, and to the region around the posterior cytopyge. This form resembles Bundle's *Bütschlia postciliata*, which differs from the more typical members of the genus in not having a complete covering of cilia.

Genus: **Isotricha** Stein, 1858.

There are two species of this genus, both of which were described by Stein (1858) from the stomach of cattle and sheep. The organisms are ovoid, with rounded anterior and more pointed posterior ends (Fig. 504, 8). The body, which is somewhat flattened, is covered with cilia, and there is a cytostome either at the anterior end or laterally placed. A large, elongate macronucleus lies longitudinally in the anterior region of the body, and close to it is a small micronucleus. There are many contractile vacuoles distributed superficially at the central part of the body. An anal aperture is present at the posterior end. The organisms are from 70 to 150 microns in length.

Genus: **Dasytricha** Schuberg, 1888.

There is a single species of this genus, which is also a parasite of the rumen of cattle and sheep, where it was first seen by Schuberg in 1888 (Fig. 504, 9). *D. ruminantium* is very similar to the species of *Isotricha*, but differs in that the body is more regularly ovoid. It is covered with cilia, and has an anterior cytostome leading to a curved œsophagus. The macronucleus is a small curved body, and near it is a micronucleus. There is a single contractile vacuole. An anal aperture occurs at the posterior end of the body.

Genus: **Didesmis** Fiorentini, 1890.

Two species of this genus were described by Fiorentini (1890) from the cæcum of the horse. They have somewhat quadrangular bodies, while at the anterior end is a round ciliated depression in which the

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1. *Butschlia parva* from the stomach of the ox (× *ca.* 1,000).
2. *B. postciliata* from the cæcum of the horse (× *ca.* 1,000).
3. *Holophryoides ovalis* from the cæcum of the horse (× 300).
4. *Blepharozoum zonatum* from the cæcum of the horse (× 175).
5. *Prorodonopsis coli* from the cæcum of the horse (× 500).
6. *Paraisotrichopsis composita* from the cæcum of the horse (× 550).
7. *Blepharoconus hemiciliatus* from the cæcum of the horse (× 300).
8. *Isotricha intestinalis* from the stomach of the ox (× 400).
9. *Dasytricha ruminantium* from the stomach of the ox (× 1,000).
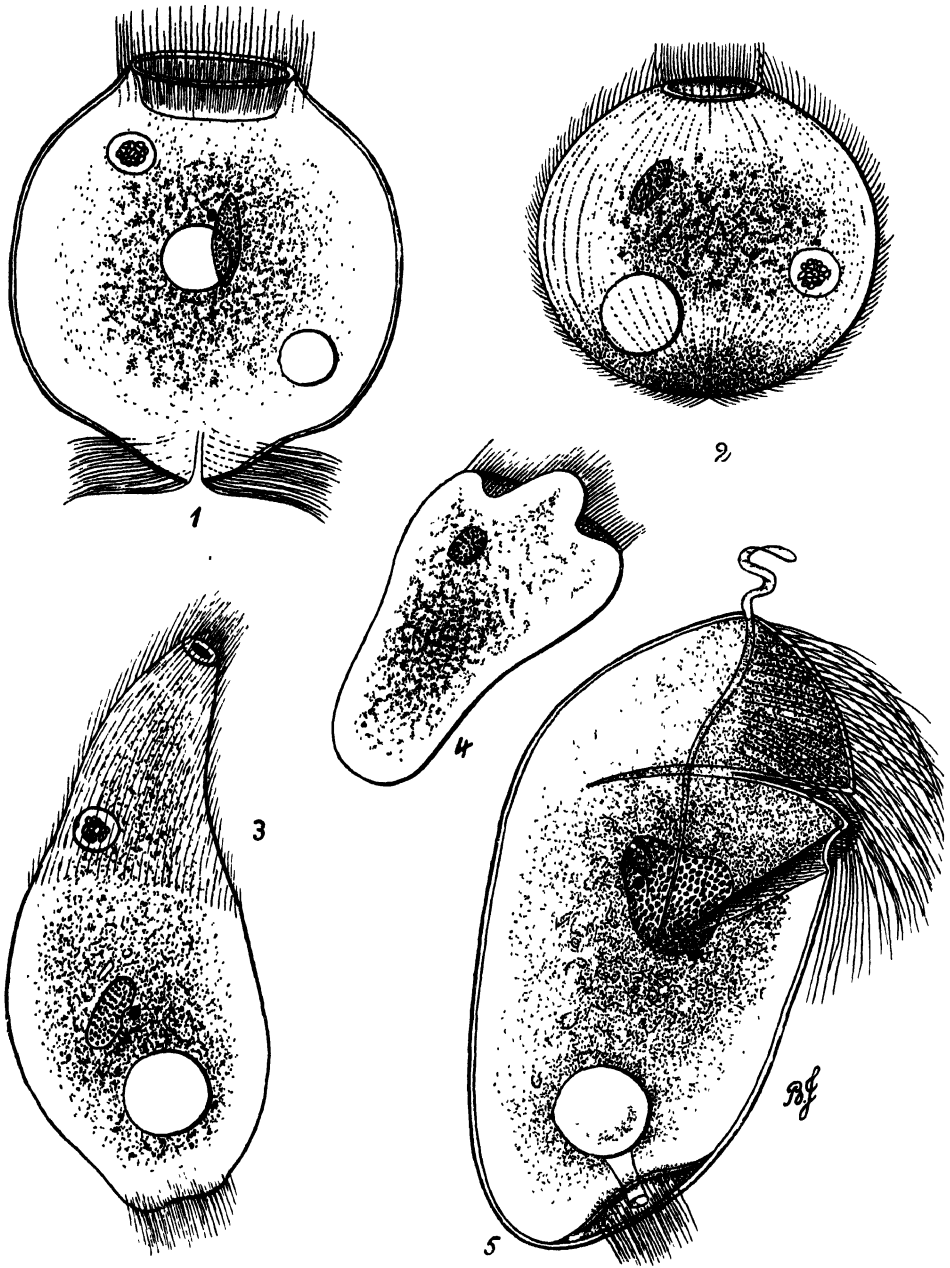
FIG. 505.—HOLOTRICHOUS CILIATES FROM THE CÆCUM OF THE HORSE.
(AFTER BUNDLE, 1895.)

1. *Didesmis ovalis* (× 2,000).
2. *Blepharosphæra intestinalis* (× ca. 500).
3. *Blepharoprosthium pireum* (× ca. 4,000).
4. *Blepharocodon appendiculatus* (× ca. 1,400).
5. *Blepharocorys uncinata* (× ca. 2,000).

cytostome occurs (Fig. 505, 1). An anal aperture surrounded by cilia is present at the posterior end. There is a macronucleus and a micronucleus, and contractile vacuoles are present.

Genus: **Blepharosphæra** Bundle, 1895.

A single species was described by Bundle (1895) from the cæcum of the horse (Fig. 505, 2). The body is spherical, and covered with longitudinally arranged rows of cilia. There is a round ciliated depression at the anterior end of the body. The macronucleus is a small structure in the anterior region, and a single contractile vacuole is present.

Genus: **Blepharoprosthium** Bundle, 1895.

A single species was described by Bundle (1895) from the cæcum of the horse (Fig. 505, 3). The body is elongate, and the anterior end is pointed and terminates in a cytostome. The posterior end of the body is rounded. There are a macronucleus and micronucleus near the centre of the body. The anterior third of the body is ciliated, also the posterior end.

Genus: **Blepharocodon** Bundle, 1895.

The single species of this genus was again described by Bundle (1895) from the cæcum of the horse (Fig. 505, 4). The body is elongate, and is broader anteriorly than posteriorly. At the centre of the ciliated anterior end is a protuberance. A rounded macronucleus and micronucleus are present.

Genus: **Blepharocorys** Bundle, 1895.

This genus was founded by Bundle (1895) to include certain ciliates seen by Fiorentini (1890) and himself in the cæcum of the horse. The body is ovoid and somewhat pointed at each end (Fig. 505, 5). A cytostome leading to an œsophagus is present on one side of the body near the anterior end, while at the posterior end is a ciliated region in the centre of which is an anal aperture. A curious band-like process projects from the anterior end. A large macronucleus and a micronucleus are present at the centre of the body. A contractile vacuole is near the posterior end. The region of the body between the cytostome and the anterior end is ciliated. Jameson (1925) has given the name *Charon ventriculi* to a ciliate from the stomach of ruminants. It resembles members of the genus *Blepharocorys*, but the anterior end is more simply organized.

Genus: **Cyathodinium** Cunha, 1914.

This genus was founded by da Cunha (1914a) for ciliates found by him in the cæcum of wild guinea-pigs (*Cavia aperea* and *C. porcella*) in Brazil. Three species were described, which differ from one another in size and in the shape of the body (Fig. 506, 1-3). In the elongate conical

form the peristome is at the anterior region of the body, while in the more globular form it is laterally situated. The cilia are limited to the region of the peristome. The macronucleus is either spherical or elongate, and a small micronucleus occurs beside it. Fantham (1925) records *C. conicum* from the guinea pig (*C. porcella*) of S. Africa.

Genus: **Paraisotricha** Fiorentini, 1890.

This genus was founded by Fiorentini (1890) for certain ciliates seen by him in the cæcum of the horse. In many respects they resemble species of *Nyctotherus*. The body is ovoid, and there is a peristome region at one side of the body, leading to a cytostome and œsophagus. The body is covered with cilia, and there is a rounded macronucleus and a
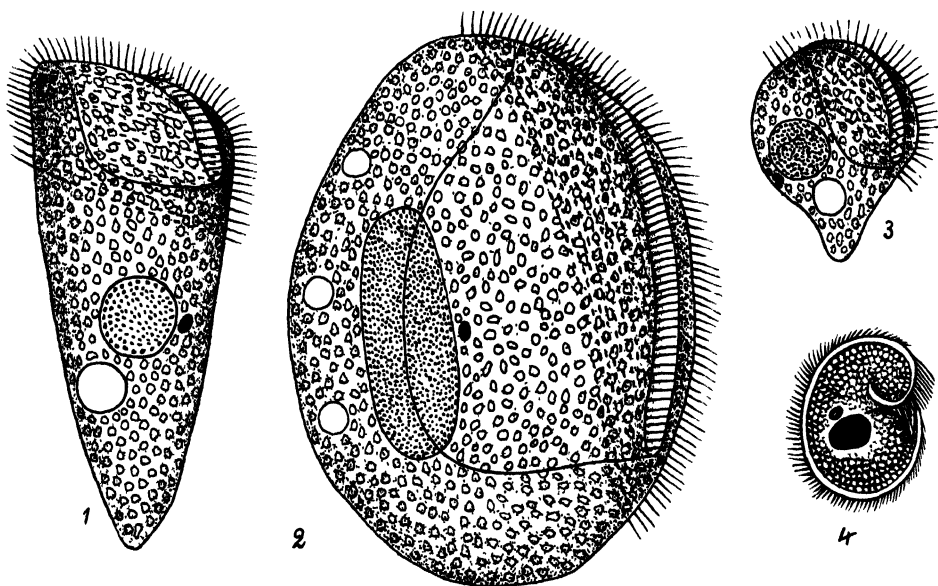
FIG. 506.—HOLOTRICHOUS CILIATES FROM THE CÆCUM OF SOUTH AMERICAN RODENTS. (AFTER DA CUNHA, 1914 AND 1915.)

1-3. *Cyathodinium conicum, C. vesiculosum,* and *C. piriforme* from the cæcum of the wild guinea-pig (× ca. 1,000). 4. *Paraisotricha hydrochœri* from the cæcum of the capibara (× ca. 500).

micronucleus. Fiorentini (1890) described six distinct species which were united into a single species by Bundle (1895). Da Cunha (1915) in Brazil recorded two species from the cæcum of the capibara, *Hydrochœrus capybara* (Fig. 506, 4).

Genus: **Enterophrya** Hasselmann, 1918.

This genus was founded by Hasselmann (1918) for certain ciliates which occurred in the cæcum of the wild guinea-pig, *Cavia aperea,* of Brazil. The two species are of a somewhat elongate piriform shape. There is

no cytostome, and cilia occur only on the anterior part of the body. A round macronucleus occurs in the posterior region, and near it is a small micronucleus. There is a single contractile vacuole near the macronucleus. A ciliated groove starting at the anterior end of the body is a characteristic feature. *E. elongata* measures 30 to 50 microns in length by 3 to 10 microns in breadth, while *E. piriforme* measures 35 by 8 microns.

Genus: **Nicollella** Chatton and Pérard, 1919.

This genus was founded by Chatton and Pérard (1919) for a ciliate of the large intestine of the gondi (*Ctenodactylus gundi*) of Tunis (Fig. 507, A).
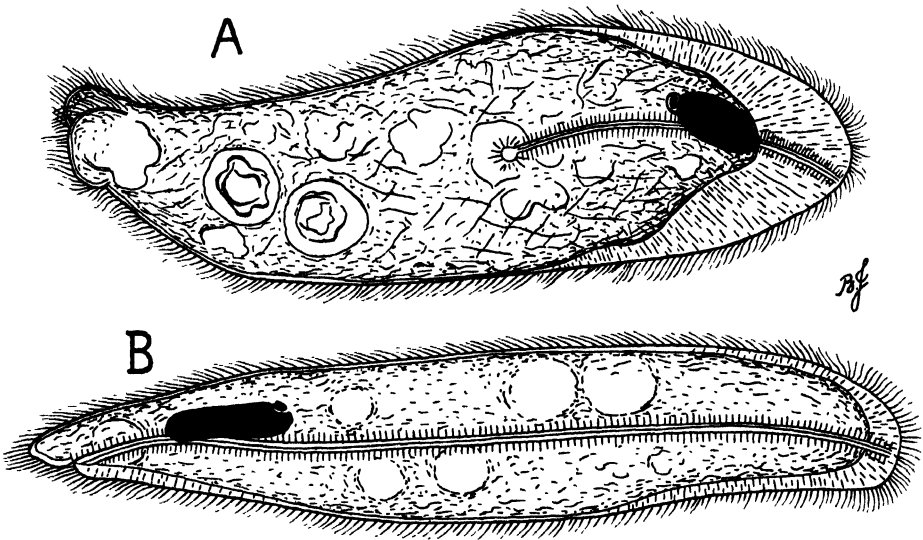
Fig. 507.—Ciliates from Intestine of the Gondi, *Ctenodactylus gundi* (× 270). (After Chatton and Pérard, 1919.)

A. *Nicollella ctenodactyli.*   B. *Collinella gundii.*

There is one species, *N. ctenodactyli*, which in its fully-grown form measures 500 to 550 by 150 microns. The organism is uniformly ciliated, and has a groove running from the anterior end to the cytostome, which is situated at the middle of the body. The posterior extremity is bi-lobed, and the contractile vacuole, which is posteriorly situated, opens between the lobes. The ovoid nucleus and micronucleus are in the anterior region of the organism. Multiplication by oblique transverse division takes place. In the material studied there occurred small individuals measuring 70 by 40 microns, many of which were in conjugation.

Genus : **Collinella** Chatton and Pérard, 1919.

Like the preceding genus, this one was established by Chatton and Pérard (1919) for a ciliate of the large intestine of the gondi (Fig. 507, B). It measures 550 to 600 microns in length, and 100 microns in breadth. The body is uniformly ciliated, and a groove passes from the anterior end of the body to the posterior end, where the cytostome is situated. The contractile vacuole is near the cytostome, while the nucleus and micronucleus are also in this region. Multiplication by transverse fission occurs. Conjugating forms were also observed. There is one species, *C. gundi,* though, according to Chatton and Pérard, Schubotz (1907) observed in the hyrax (*Procavia capensis*) of South Africa another ciliate which evidently belongs to this genus.
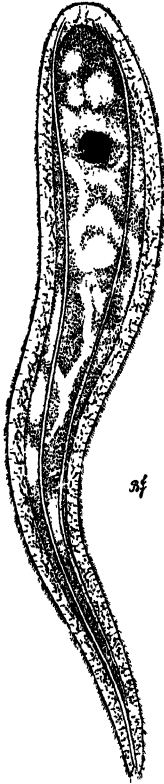
Genus : **Pycnothrix** Schubotz, 1907.

This genus was founded by Schubotz (1907) for a large ciliate named by him *Pycnothrix monocystoides,* which he found in the large intestine of the South African hyrax, *Procavia capensis* (Fig. 508). According to Schubotz, it may reach a length of 3,000 microns and a breadth of 500 microns. The nucleus is a spherical or ovoid body in the anterior region of the ciliate. Sometimes a small mass situated in a depression of the nucleus can be detected. This has been interpreted as the micronucleus. Schubotz described two grooves which pass along each side of the body and terminate posteriorly. Chatton and Pérard (1921) pointed out that these two grooves commence one on each surface of the anterior end of the somewhat flattened body, and are continuous at the notched posterior end. In the grooves are a series of openings which lead into the endoplasm. These are regarded as buccal orifices. At the junction of the middle and posterior thirds of the body is a pore which lies to the side of one of the grooves. It communicates with a system of branching canals which ramify in the endo-

FIG. 508.—*Pycnothrix monocystoides* FROM THE INTESTINE OF *Procavia brucei* (× 45). (AFTER CHATTON AND PÉRARD, 1921.)

The two grooves, one on each surface of the body, are visible. In these grooves occur various openings, which are regarded as cytostomes.

plasm. The canals, which are ciliated near the orifice, are regarded as an excretory system corresponding with a highly developed contractile vacuole. The ectoplasm of the organism is thick, and consists of three distinct layers. The writer examined some of Schubotz' material and found that the organisms present varied in length from 300 to 2,000 microns. In the majority of the fixed forms the cilia had disappeared and it was only in a small number that anything like a micronucleus could be detected. According to Chatton and Pérard (1921), the organism was seen by Brumpt in the intestine of *Procavia brucei* of Abyssinia, and it was this material which was studied by them.

2. *SUB-CLASS*: Spirigera.

(1). *Order*: HETEROTRICHIDA DELAGE AND HÉROUARD, 1896.

The typical ciliates included in this order (=Heterotricha Stein, 1859) are covered with cilia, and possess an adoral zone of spirally arranged cilia which are longer than those on the body and lead to the cytostome. As in the preceding order, the shape of the body varies considerably. The species of *Spirostomum*, for instance, are elongate, almost worm-like ciliates which are easily visible to the naked eye (Fig. 509). They are often seen as white threads in stagnant water, and may reach a length of 4 or 5 millimetres. The adoral zone of long cilia and the cytostome are laterally placed at one end of the body, while at the other is a large contractile vacuole. There is an elongate beaded macro-nucleus, which is nearly as long as the body and several micro-nuclei. Other free-living forms belonging to this order are more nearly ovoid in shape, while *Stentor* is cone-shaped (Fig. 22). Several species belonging to the genera *Nyctotherus* and *Balantidium* are parasites of the intestinal tract of man and animals.
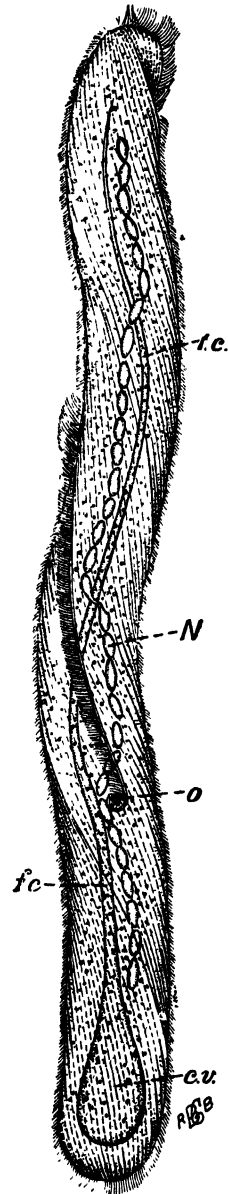
FIG. 509.—*Spirostomum ambiguum*. (× 185). (FROM MINCHIN, 1912, AFTER STEIN.)

N, Macronucleus; *o*, mouth at posterior end of long peristome; *c.v.*, contractile vacuole at end of long canal (*f.c.*).

Genus : Nyctotherus Leidy, 1849.

The ciliates belonging to this genus, one species of which has been recorded as a parasite of man, are flattened dorso-ventrally and kidney-shaped in outline (Fig. 511). There is a notch near the middle of the right side of the body which is covered with cilia, and there is an adoral zone of cilia on the peristome in front of the notch leading to the cytostome, an opening situated in the notch. The cytostome is continuous, with a long, curved œsophagus, on the anterior wall of which is a row of parallel plates of fused cilia. This row of plates extends in the adoral region nearly as far as the anterior end of the body. The genus was founded by Leidy (1849) for a parasite of the myriapod, *Julus marginatus,* to which he gave the name *Nyctotherus velox.*

NYCTOTHERUS IN MAN.

Nyctotherus faba Schaudinn, 1899.—This form was described by Schaudinn (v. Jakoby and Schaudinn, 1899) from a case of diarrhœa in a man who had come from America. Another ciliate named by Schaudinn, *Balantidium minutum,* was present in the same stool. *N. faba* is 26 to 28 microns in length, 16 to 18 microns in breadth, and 10 to 12 microns in thickness (Fig. 510). The notch or peristome is on the right side of the body near the anterior end, and the cytostome leading to the short œsophagus is situated at the posterior end of the peristome at the middle of the body. At the centre of the body is a large spherical macronucleus, near which lies the small micronucleus. A large contractile vacuole is present at the posterior region of the organism. Encysted forms were also described as ovoid bodies, presumably of the same dimensions as the free forms. Sangiorgi and Ugdulena (1917) saw a ciliate in the stools of a man in Italy which they ascribe to this species, while Pinto (1919) claims to have seen the ciliate in Brazil. The organism is probably non-pathogenic, and it is very doubtful if it belongs to the genus *Nyctotherus.* The possibility of it being a free-living form does not appear to have been excluded.

FIG. 510.—*Nyctotherus faba* FROM HUMAN FÆCES (× *ca.* 1700). (AFTER SCHAUDINN, 1899.)

Krause (1906) observed in the stool of a typhoid patient a large ciliate, which he named *Balantidium coli giganteum.* Braun (1908) placed it in the genus *Nyctotherus* as *N. giganteum,* as did also Doflein (1916). The

organism varied in length from 60 to 400 microns, and in breadth from 60 to 150 microns. Encysted forms were also described. Apart from the fact that the shape of the body resembled that of *Nyctotherus*, there is nothing to justify its inclusion in this genus. It is only possible to conjecture that it may have been an abnormally large form of the free-living *Colpoda cucullus*.

Castellani (1905) described a ciliate of an hour-glass shape which he saw in the stool of a case of sleeping sickness in Uganda. He gave it the name *N. africanus*, but there is nothing in the description to warrant its inclusion in this genus, or indeed in any other known genus. The shape would suggest that it was possibly a dividing form of a *Balantidium*. Guérin (1926) states that he has rediscovered *N. africanus* in the fæces of a soldier in E. Africa. It is not clear from his account that the ciliate belongs to this genus.

NYCTOTHERUS IN ANIMALS.

Nyctotherus cordiformis (Ehrenberg, 1838).—This ciliate is a very common parasite of the rectum of the frog, where it is nearly always found in association with *Opalina ranarum* and *Balantidium entozoon*. According to Dobell (1909), it was probably first seen by Leeuwenhoek (1683, 1702), but the first clear account was given by Ehrenberg (1838), who described it under the name *Bursaria cordiformis*. It is the most readily obtained member of the genus, and occurs in *Rana temporaria*, *R. esculenta*, *Bufo cinereus*, *Bombinator igneus*, *Hyla arborea*, and possibly other frogs and toads.

As usually seen, *N. cordiformis* varies in length from 60 to 120 microns, and is about half this in breadth (Fig. 511). There is a long œsophagus, which passes into the cytoplasm from the cytostome at the middle of the right side of the body. The œsophagus, which has a series of parallel plates of fused cilia, curves in a backward direction towards the posterior portion of the body. In front of the cytostome is the peristome, a slightly flattened area on which are the long adoral cilia and a continuation of the plates which occur in the œsophagus. The macronucleus is a sausage-shaped, curved body anterior to the œsophagus, and a small micro-nucleus lies on its concave side. There is a contractile vacuole at the posterior end of the body, near which is the anal aperture. The ciliate multiplies in the usual manner by binary fission, and at certain times encystment occurs. The cyst, which was first described and figured by Stein (1867), is an ovoid structure 80 to 90 microns in length, and contains a single ciliate. There is no indication that the organism is in any way pathogenic.

N. macropharyngeus, measuring 350 by 200 microns, and *N. magna*,

660 by 460 microns, are two forms described by Bezzenberger (1904) from Asiatic frogs (*Rana tigrina*, *R. hexadactyla*, and *R. cyanophlyctis*). *N. macropharyngeus* was seen by Dobell (1910) in *R. tigrina* in Ceylon, as also a new species, *N. papillatus*, in *Bufo melanostictus* and *Rhacophorus maculatus*, of the same locality.

N. ovalis was the name given by Leidy (1849, 1850) to a form which occurs in the intestine of the common cockroach, *Blatta orientalis* and
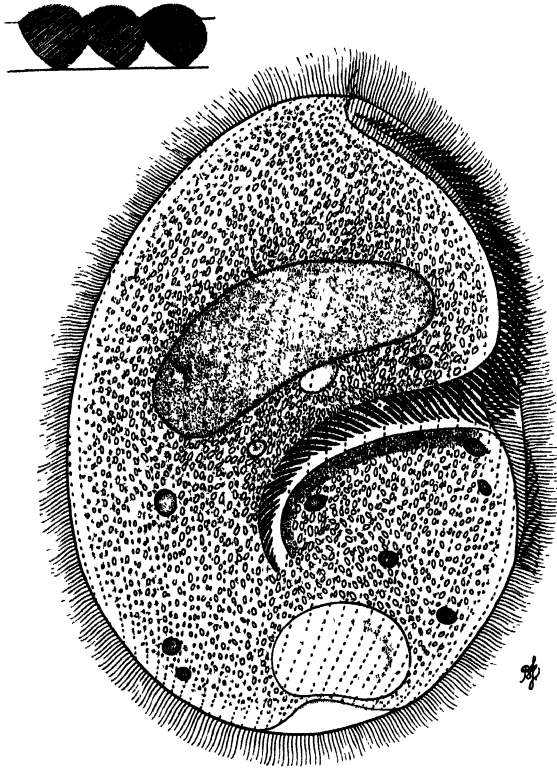
FIG. 511.—*Nyctotherus cordiformis* FROM THE RECTUM OF THE FROG (× 500). (ORIGINAL.)

Three of the plates of adherent cilia which occur in the cytopharynx are shown at the side.

B. germanica, and the mole cricket, *Gryllotalpa vulgaris*. He also saw another form, *N. velox*, in the myriapod, *Julus marginatus*. Daday (1905) and Entz (1913a) have described *N. piscicola* from the intestine of certain fresh-water fish of South America. *N. termitis* is a form described by Dobell (1910) from white ants, *Calotermes militaris*, of Ceylon.

Other species of *Nyctotherus* have been described, and it is evidently a common intestinal parasite.

Genus : **Balantidium Claparède and Lachmann, 1858.**

This genus was founded by Claparède and Lachmann for a ciliate which Ehrenberg (1838) had observed in the rectum of frogs, and which he had named *Bursaria entozoon*. The members of this genus, which include species in a variety of hosts, both vertebrate and invertebrate, have pear-shaped bodies completely covered by longitudinal spirally arranged rows of cilia (Fig. 512). At the anterior end of the ventral surface is the peristome, a depressed area the anterior or broader end of which is at the anterior end of the body, while its narrow posterior end is on the ventral surface. The cytostome is an opening in the depression of the peristome, and from it extends the œsophagus, which ends blindly in the endoplasm. Commencing at the posterior end of the margin of the peristome is an adoral row of long cilia. This row passes along the right margin of the peristome across its anterior margin, and then backwards along the left margin to a point near which the adoral row started. It then passes into the cytostome, and is continued backwards in the same spiral manner till it reaches a point half-way down the œsophagus on its ventral wall, where it ends. The longitudinal rows of cilia on the ventral surface also pass over the posterior margin of the peristome, and are continued along the ventral wall of the œsophagus till they meet the row of adoral cilia in its spiral course. It seems probable that the longitudinal rows of cilia on the ventral wall of the œsophagus are fused to form a number of membranes. At the posterior end of the body is an anal aperture or cytopyge. There are one or more contractile vacuoles. The macronucleus may be sausage-shaped or spherical, while a small micronucleus is closely applied to it.

BALANTIDIUM IN MAN.

A large number of species of *Balantidium* has been described, especially from Batrachians. Two species occur in pigs, and one of these is found also in human beings, who are liable to infection with two species.

Balantidium coli (Malmsten, 1857).—This ciliate was first discovered by Malmsten (1857) in two patients suffering from dysentery. He referred to it as *Paramecium (?) coli*. Claparède and Lachmann (1858) transferred it to the genus *Plagiostoma*, while Stein (1860) regarded it as belonging to the genus *Leucophrya*. Leuckart (1861a) discovered a ciliate in the intestine of pigs, and considered it as identical with the human form. He suggested placing both of them in the genus *Holophrya*. Stein (1863) came to the conclusion that they belonged to the same genus as *Balantidium entozoon* of the frog, and renamed them *Balantidium coli*, by which name they are now known.

Dobell (1920) points out that the discovery of the ciliate in man is often ascribed to Leeuwenhoek, owing to a misconception in regard to the flagellate *Giardia intestinalis*, which he was actually describing. Leuckart (1861*a*, 1863) found that the organism was very commonly present in the intestine of pigs, an observation which was confirmed by Stein (1863) in Germany, Wising (1871) in Sweden, Grassi (1881*a*) in Italy, and by many other observers. These animals probably harbour the parasite in all parts of the world. Brooks (1903) discovered *B. coli* in orang-outangs in New York, and Noc (1908) and Brumpt (1909) in species of *Macacus*. One of these monkeys was found infected by Stevenson in London. Joyeux (1913) observed a species of *Balantidium* in the baboon (*Papio sphinx*) of French Guinea, and was able to infect another baboon from this one. Hegner (1923) in North America has noted an infection in *Cebus variegatus* which came from Brazil. Christeller (1922) found *B. coli* in the intestine of two chimpanzees which had died of dysentery in the Zoological Gardens in Berlin. The mucosa of the large intestine was ulcerated, and contained the ciliates. As no other cause of the condition could be found, the apes were assumed to have died of balantidial dysentery. Ziemann (1925) records infections of man, chimpanzees, and *Cercocœbus fuliginosus* in Germany. Da Cunha (1917) saw a *Balantidium* in a horse in Brazil, and noted its resemblance to *B. coli*.

Distribution.—*B. coli* is widely distributed throughout the world, and has been recorded from various parts of Europe, Asia, Africa, and America. In human beings, infections are most common in individuals who come into association with pigs, and there is every reason to believe that the ciliate, which is a common parasite of the pig, is the same as that which occasionally infects man and monkeys. Walker (1913) points out that, in the fifty-six years following Malmsten's first case in 1857, only 137 cases of human infection have been recorded. In the Philippines alone fifty-seven cases had come under observation.

Morphology.—*B. coli* is shaped like an egg or pear (Fig. 512). It measures, as a rule, from 50 to 80 microns in length, and has a breadth of a little more than two-thirds of its length. Larger and smaller forms have been described, and Brumpt gives the measurements as varying from 30 to 200 microns by 20 to 70 microns, while McDonald (1922) gives a length of 30 to 150 microns and a breadth of 25 to 120 microns. At the anterior end, and arranged somewhat obliquely, is a depression, the peristome, which is actually on the ventral surface of the ciliate. With the constant changes in shape of the anterior end of the body, the appearance of the peristome varies. Sometimes it is a wide-open depression; at others a longitudinal groove or slit. On its margins is the adoral zone of cilia, which pass round it, and eventually through the cytostome into

the œsophagus, as described above. According to McDonald (1922), the unciliated cytoplasm between the adoral cilia and the lip of the cytostome may form a kind of plug, the oral plug, which can completely close the opening of the cytostome. The whole body is covered with cilia arranged in longitudinal rows in the grooves between the ectoplasmic ridges. These rows take a slightly spiral course in such a way that a row which starts towards the left of the anterior end of the body passes over the dorsal surface towards the right of the posterior end. The cilia on the body are
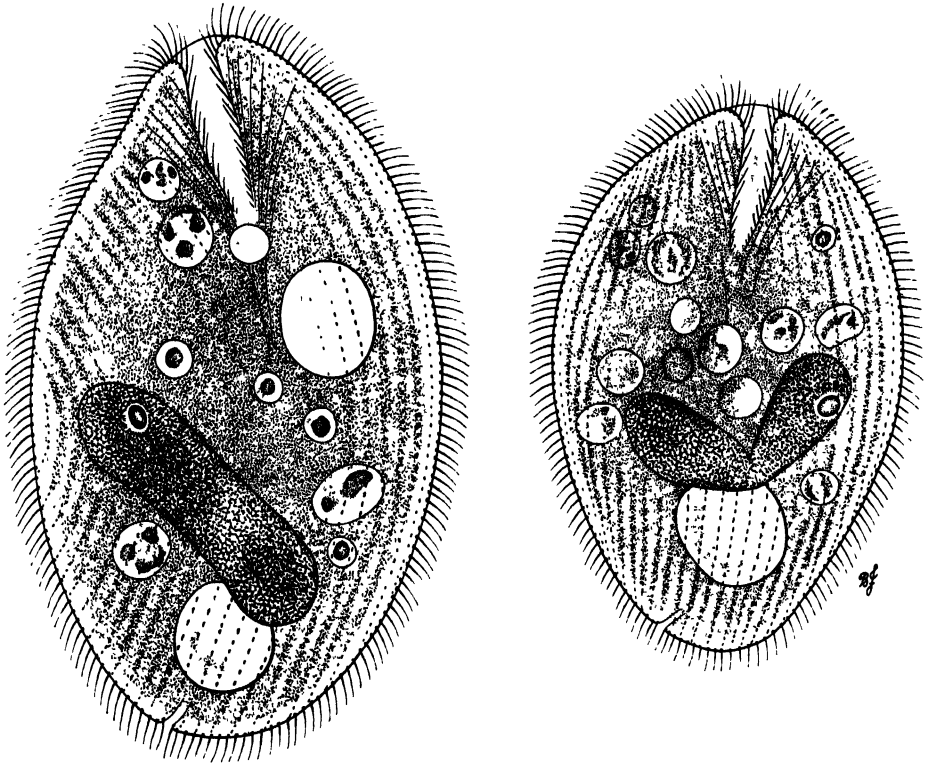
FIG. 512.—*Balantidium coli* FROM THE HUMAN INTESTINE (× 1,200). (ORIGINAL.)

stated by McDonald to be 4 to 6 microns in length, while those of the adoral zone measure 8 to 12 microns. A sausage-shaped macronucleus lies more or less transversely at the middle of the body, while, close to it, and often in a slight depression, is the small micronucleus. There are two contractile vacuoles, one at the posterior end and the other near the middle of the body. An anal aperture is present at the posterior end. The surface of the body, which is longitudinally ridged, is limited externally by a fine pellicle, beneath which is a clear ectoplasm. The appearance of the endoplasm varies with the state of nutrition.

Sometimes there are numerous vacuoles, each of which contains a highly refractile globule. At other times the vacuoles contain red blood-corpuscles, leucocytes, or other débris. McDonald (1922) has described in association with the cytostome and adoral cilia a neuromotor system of fibres and a co-ordinating centre or motorium, which is embedded in the ectoplasm near the œsophagus. Following other workers of the Californian school, such as Sharp (1914), Yocom (1918), and Taylor (1920), he supposes that this system regulates and co-ordinates the movements of the adoral cilia and the peristome region of the body (see p. 120).

Reproduction is by transverse fission after division of the two nuclei. In this process the body becomes constricted and hour-glass shaped. The cytostome remains with the anterior individual, while a new cytostome is formed for the posterior one. Multiplication takes place in this manner both in the lumen of the gut and in the tissues in those cases in which the ciliate has penetrated the mucosa.
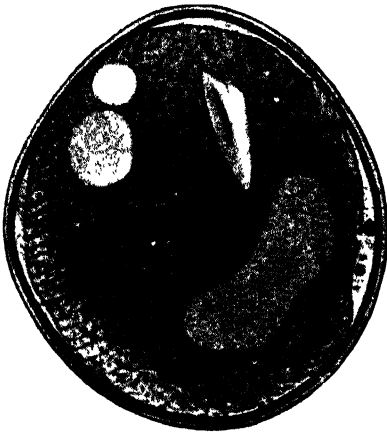
FIG. 513.—*Balantidium coli:* EN-CYSTED FORM, AS SEEN IN LIVING CONDITION IN FÆCES OF PIG (× 1,000). (AFTER DOBELL AND O'CONNOR, 1921.)

The elongate macronucleus, two vacuoles, and an angular inclusion body are visible.

Walker (1909) described a process of sporulation as occurring in the tissues of man. Nests of small ciliates occur, and Walker believes that these arise by a process of multiple segmentation of a large one. It would seem more probable that they had resulted from repeated and rapid binary fissions, and not by a process which is not known to occur amongst the Ciliata.

Conjugating stages have been described, and were first depicted by Leuckart. According to Brumpt (1909, 1913c), two ciliates become attached to one another by their peristomes and enclosed in a cyst. The exact details of conjugation within the cyst have not been worked out, but it is supposed that the bodies of the two ciliates fuse. More frequently, single individuals become encysted. In this process a ciliate becomes more nearly spherical, and secretes around itself a tough cyst wall, which consists of two distinct layers (Fig. 513). The cyst appears to be a purely protective structure, for no multiplication has been seen to occur within it. The ciliate can be seen inside with its cilia in slow movement. The cysts measure from 50 to 60 microns in length, and slightly less than this in breadth. They are voided in the fæces, and undoubtedly serve to spread infection.

Pathogenicity.—Though the majority of cases of *B. coli* infection which have been recorded have occurred in subjects suffering from diarrhœa, this by no means indicates that the ciliate was the cause of the trouble. As Walker (1913) points out, the physician's attention is only drawn to a stool when there is something abnormal. The stools of apparently healthy people are not examined. Walker found that of fifty-seven cases of infection in the Philippines, only eleven showed diarrhœic or dysenteric symptoms. It appears that *B. coli* behaves very much like *Entamœba histolytica* in this respect. It is only a small proportion of infected individuals who are so seriously upset that definite symptoms are produced. The mortality in 111 cases, according to Strong (1904), was 29 per cent. In the fifty-seven Philippine cases, Walker gives the mortality as 7 per cent., but states that most of these were latent cases without symptoms. He points out that at forty *post-mortem* examinations recorded in the literature, ulceration of the large intestine was present in thirty-six. It has been noted above that in the case of *E. histolytica* infections, it is probable that there is always ulceration of the large intestine, even in carriers without symptoms. It has also been demonstrated that persons infected with *B. coli* may have extensive ulceration of the gut without any symptoms having been noted, so that it may be suspected that infections of man with *B. coli* are always associated with ulceration, as in the case of *E. histolytica*.

Pathology.—The pathology of balantidiosis has chiefly been studied in the Philippines by Strong (1904), Bowman (1909, 1911), Walker (1913), and Manlove (1917), and in France by Brumpt (1913c). The ulceration produced by *B. coli* is usually limited to the large intestine, though, as in the case of *E. histolytica*, ulceration of the small intestine may occasionally occur. Thus Reis (1923) has found that in four cases of balantidial dysentery of man investigated by him, the ciliates were present in the lower part of the small intestine and also in the large intestine.

The appearance of the large intestine is very similar to that seen in amœbic dysentery, and the two conditions can only be differentiated by the discovery of their respective parasites (Fig. 514). The ulcers have undetermined edges, and the ciliates are found in the cavity of the ulcer and also in the tissues surrounding it. They occur in the submucosa, muscularis, blood-vessels, and lymph spaces. They are also found in the lymphatic glands draining the infected area of the gut, but they do not occur in the liver, though Stokvis (1884) recorded the presence of a ciliate which he regarded as *B. coli* in material coughed up from the lung. It was presumed that a liver abscess had ruptured into the lung. In the lesions there is a tendency for the ciliates to be arranged in groups. They can be recognized in sections as compact bodies 50 to 80 microns in length, with

large sausage-shaped macronuclei which stain intensely with hæmatoxylin and other stains. Careful observation will show the cilia, cytostome, and other features of the parasite, so that there should be no difficulty in recognizing them in sections. Walker states that they enter the mucosa in the first place in a mechanical manner by forcing their way into the tissues through the epithelium between the gland openings, and not after entering the glands. They penetrate deeply and multiply by binary fission. A
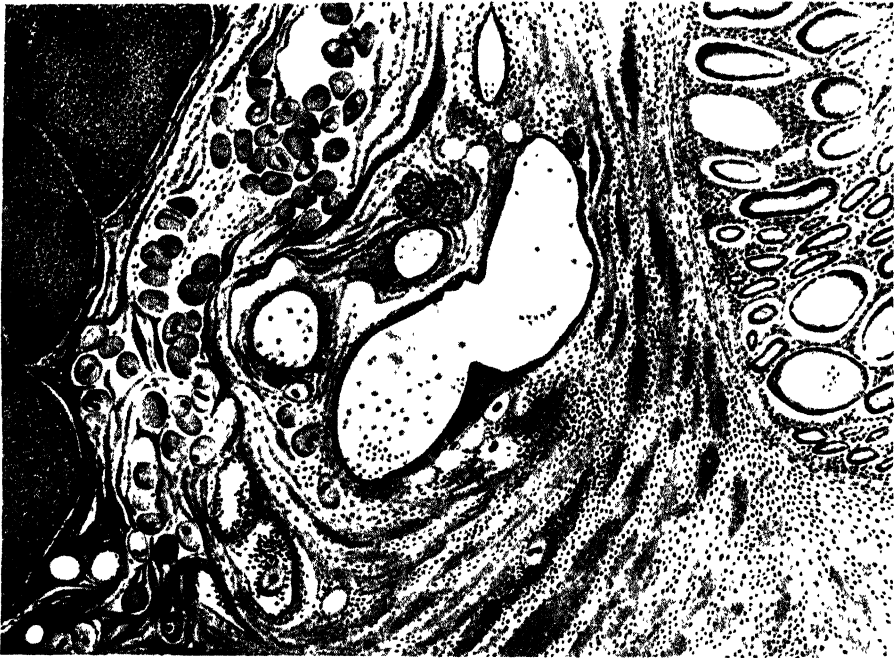
FIG. 514.—BALANTIDIAL ULCERATION OF THE LARGE INTESTINE OF MAN (× 780)
(ORIGINAL.)

Section through the margin of a large ulcer, showing *Balantidium coli* extending in the submucosa into regions where the superficial mucosa is still intact. The limit of the ulcer is where the mucosa commences at the lower part of the right-hand side of the drawing. The muscular layers of the intestine are on the left (× 780).

necrosis of the tissues is produced, and eventually an abscess is formed, which bursts into the lumen of the intestine and gives rise to the open ulcer.

Marshall (1911a) stated that he had cultivated an organism like *B. coli* while attempting to grow *Leishmania donovani* from a case of kala-azar *post-mortem* in the Sudan. Maliwa and Haus (1920) claimed to have seen it in the urine of a woman, and to have discovered it also in the bladder and ureter after death. Hinkelmann (1919) stated that

he had cultivated the ciliate from the peripheral blood of a case which showed it in the urine. It is difficult to account for these records unless it be assumed that the supposed *B. coli* were free-living ciliates which had contaminated the materials examined.

Susceptibility of Animals.—As already pointed out, *B. coli* occurs in the pig and monkey. Wising (1885), Grassi (1888*a*), and others have held the view that the ciliate of the pig is a distinct species, but the experimental evidence adduced by Brumpt (1909) and Walker (1913), and the fact that a large proportion of infected human beings are known to have been in a position to acquire their infections from pigs, makes the identity of the two almost certain.

Brumpt (1909) experimented with the ciliates of the pig, and those seen in six monkeys (*Macacus cynomolgus*) which had come to France from Indo-China. It was demonstrated that the ciliates of the monkey could be handed on to other monkeys, and also to young pigs. It was also shown that the naturally occurring ciliate of the pig could be inoculated to monkeys. Walker (1913) published accounts of further experiments. Of five monkeys, either fed or injected *per rectum* with *B. coli* from human cases, two became infected. Furthermore, of seventeen monkeys similarly treated with *B. coli* from the pig, twelve became infected. Only three of these monkeys showed *B. coli* in the tissues at *post-mortem* examination. It thus appears evident that human beings, monkeys, and pigs, as hosts of *B. coli*, are interchangeable. Several observers have, however, failed to produce infections in monkeys and pigs with *B. coli* of human origin, as also in rabbits and dogs. It is generally supposed that *B. coli* in pigs does not cause any lesions of the gut, but in one of the pigs infected by Brumpt there was diarrhœa associated with blood and mucus, and the ciliates in the stool contained red blood-corpuscles. At autopsy, lesions of the large intestine were found. The monkeys infected by Brumpt showed no gut lesions, but these were present in Walker's experimental infections. In the naturally occurring case seen by Stevenson in a *Macacus* in London, there was no evident ulceration. In sections of the gut, however, the ciliates were seen in places to be closely applied to the mucosa, or even sunk in depressions of its surface.

Culture.—Most of the attempts which have been made to cultivate *B. coli* have been unsuccessful. Barret and Yarbrough (1921), however, have reported the successful culture of the ciliate for over thirty-two days, during which eleven subcultures were made. The medium employed was a mixture of one part of inactivated human serum and sixteen parts of 0·5 per cent. salt solution, the mixture being faintly alkaline to litmus. About 0·1 c.c. of the undiluted fæces containing the ciliates was inoculated

by means of a pipette into the medium at the bottom of test-tubes (1 × 15 cm.) containing 8 c.c. of the medium. The tubes were then incubated at 37° C., and examined after twenty-four hours by abstracting fluid from the deeper portions by means of a pipette. At this time there was only moderate growth, but the optimum occurred after forty-eight or seventy-two hours. As a rule subculture had to be made every two days. In the cultures the ciliates multiplied by binary fission. Encysted forms were occasionally seen, and on one occasion what was apparently a conjugating pair.

The organism described by Castellani (1914, 1914a) as *Entoplasma* (*E. castellanii* Paul, 1914) is probably a badly fixed *B. coli*. The writer examined the preparations, which were dried films of the fæces stained by Romanowsky stain. It was impossible to identify the organism with certainty, but from the fact that there was some indication of an anterior cytostome, the probability is that it was *B. coli*, with the dimensions of which it conformed.
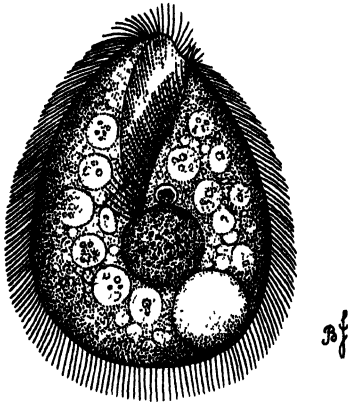
FIG. 515.—*Balantidium minutum* FROM HUMAN FÆCES (× 1,700). (AFTER SCHAUDINN, 1899.)

Action of Drugs on B. coli.—The long list of medicaments which have been used in the treatment of balantidiosis of man is in itself an indication that no specific drug has yet been discovered. As was clearly pointed out by Walker, *B. coli* may vanish from the stool of infected individuals for considerable periods and reappear later. The actual presence of ciliates in the stool is thus subject to a periodicity, and in judging the action of any remedy which has been given this fact must be kept in mind. The good results which have been reported are probably explicable on these grounds. Treatment by the mouth with ipecacuanha, emetin, carbolic acid, thymol, male fern, and high rectal irrigation with solutions of quinine, silver nitrate, salicylic acid, and iodine, have all been recommended, but it is doubtful if any of these has had a beneficial effect in ridding the host of the infection.

Reis (1923) has noted that in cases studied by him the bacteria were of the acid-producing type, the alkali-producing forms being suppressed. This fact suggests to him a line of treatment based on the introduction of the latter group of organisms and the use of alkalis.

Balantidium minutum Schaudinn, 1899.—This small ciliate was described by Schaudinn (v. Jakoby and Schaudinn, 1899) from the case in

which *Nyctotherus faba* was found (see p. 1198). The body is piriform, and measures 20 to 30 microns by 14 to 20 microns (Fig. 515). The anterior end is relatively more pointed, and the cilia which cover the body longer than in *B. coli*. The peristome, which is a slit extending backwards from the anterior extremity for about half the length of the body, is well developed, and lined with cilia. At its posterior end it passes into the œsophagus. An undulating membrane is also described as occurring on
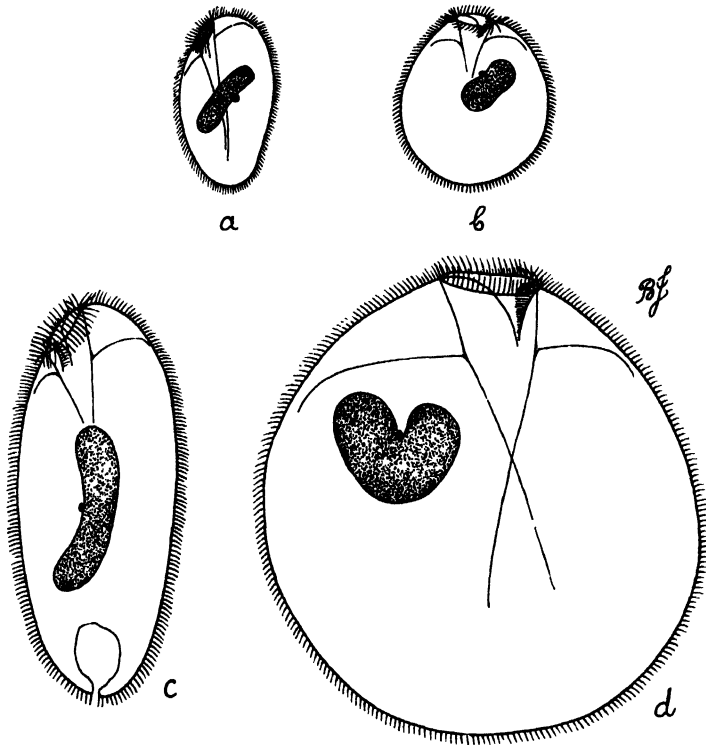
FIG. 516.—*Balantidium* OF THE PIG (× *ca.* 500). (AFTER McDONALD, 1922.)

a and c. Small and large forms of *Balantidium suis*.
b and d. Small and large forms of *Balantidium coli*.

the peristome. It is not clearly reproduced in Schaudinn's figure, and is usually neglected in accounts of this organism. One contractile vacuole is present at the hinder end of the body. The macronucleus is a spherical structure 6 to 7 microns in diameter, and by its side is a small micronucleus. Multiplication is by transverse fission, as in *B. coli*, and cysts which are ovoid in shape are described. Sangiorgi and Ugdulena (1917) claim to have rediscovered a ciliate of this type in Italy. They regard their parasite as a variety (*italicum*) of *B. minutum*. From the

description and a figure given later by Sangiorgi (1919) it seems doubtful if the ciliate belongs to the genus *Balantidium*, and this is borne out by the fact that it was easily cultivated in peptone water. It may be merely a free-living organism. Pinto (1919) has, however, recorded *B. minutum* from Brazil. It is not improbable that the ciliate is a coprozoic organism, as, indeed, Brumpt (1922) has suggested.

McDonald (1922), who has investigated *Balantidium* of pigs in America, has come to the conclusion that these animals harbour two species (Fig. 516). One of these is *B. coli*, which also infects man; the other is named *B. suis*. It is not so broad as *B. coli*, and has a length approximately double its breadth, since it measures from 35 to 120 by 20 to 60 microns. It is relatively blunter anteriorly and more pointed posteriorly than *B. coli*. The broadest part of the body is anterior to the middle point of the longitudinal axis. The nucleus is longer and narrower than that of *B. coli*, and is at least one-half the length of the body. There is no evidence that *B. suis* infects man.

BALANTIDIUM IN ANIMALS.

Apart from the ciliates which occur in monkeys and pigs, and which are probably identical with *B. coli* of man, other species are commonly seen in animals, especially frogs and toads. The form seen by da Cunha (1917) in the horse, and which he regarded as *B. coli*, has been referred to above, while *B. caviæ* was described by Neiva, da Cunha and Travassos (1914) from the guinea-pig (*Cavia aperea*) in Brazil. Hegner (1924) has seen a form in the sheep. It measured 40 to 49 microns in length by 29 to 36 microns in breadth. The writer has seen cysts which Sheather (1923) discovered in the fæces of cattle. There appears to be little doubt that they belong to a species of *Balantidium*. Frogs and other Batrachia are commonly infected with *Balantidium* (Fig. 14). Ehrenberg (1838) discovered *B. entozoon* in *Rana temporaria* and *R. esculenta*. Stein (1863) described *B. duodeni* from *R. esculenta* and *B. elongatum* from *R. temporaria*, *R. esculenta*, *Triton cristatus*, *T. alpestris*, and *T. marmoratus*. As frogs are nearly always infected, their rectal contents afford good material for the study of these ciliates. Bezzenberger (1904) described other species from Asiatic frogs. *B. helenæ* occurs in *Rana tigrini*, *R. limnocharis*, *R. cyanophlyctis*, and *R. hexadactyla; B. rotundum*, and *B. giganteum* in *R. esculenta* var. *chinensis; B. gracilis* in *R. cyanophlyctis* and *R. hexadactyla*. Dobell (1910) recorded two new species from *R. tigrina* of Ceylon (*B. ovale* and *B. hyalinum*). In hosts other than Batrachia species of *Balantidium* have also been described. *B. viride* was recorded by Willach (1893) from a bird, *Columba* sp. ?

B. piscicola from the gut of the South American fish, *Piarectus brachy-pomus* was named by Entz (1913*a*). Gauthier (1920) gave the name *Balantidium granulosum* to a ciliate from the stomach of a salmon (*Salvelinus fontinalis*).

A form seen by Chagas (1911) in the tortoise, *Testudo græca*, in Brazil, was named by him *B. testudinis*. The writer has seen this form in *T. radiata* and *T. calcarata*. *B. medusarum* was recorded by Mereschkowsky (1879) from species of *Bougainvillea*, *Obelia*, *Eucope*, and *Brada; B. amphictenides* by Entz (1888) from *Amphictenis* sp. and *Turbellaria marina; B. gyrans* by Kellicott (1888) from an aquatic worm; *B. littorinæ* by Chagas (1911) from *Littorina* sp.; and *B. hydræ* by Entz (1913*a*) from *Hydra olygactis*. Watson (1916) described as *B. orchestium* a ciliate occurring in the gut of American sand fleas, *Orchestia agilis* and *Talorchestia longicornis;* and Leon (1919) *B. haughwouti* from a Philippine snail (*Ampullaria*). Ghosh (1922, 1922*a*) in India has described two species (*B. blattarum* and *B. ovatum*) from the cockroach, *Blatta americana*. As the last-named species was founded on a single specimen only, it must be doubtful if two species are represented. This observer (1925) gives the name *Balantidium knowlesi* to a ciliate found in the cœlomic cavity of *Culicoides peregrinus* of Calcutta.

These numerous species of *Balantidium* differ from one another in the shape and size of the body, the character of the cytostome, the shape of the macronucleus, and the appearance of the cytoplasm and other details. It is very probable that some of them do not belong to the genus *Balantidium*. There is no evidence that they are in any way pathogenic, though the forms found in the frog will ingest red blood-corpuscles if these are present in the gut.

(2). *Order :* OLIGOTRICHIDA.

The ciliates belonging to this order (=Oligotricha Bütschli, 1889) do not have a uniform coating of cilia covering the whole body. The cilia are limited to special areas, and there is present an adoral zone of cilia arranged as a left-handed spiral on the peristome. There is a number of free-living forms, but many of the ciliates which live in the stomach of ruminants and in the cæcum of horses belong to this order, as also certain forms which are intestinal parasites of the chimpanzee and gorilla, and certain small rodents of South America.

Genus : Entodinium Stein, 1858.

This genus was established by Stein (1858) for several species of ciliate occurring in the stomach of ruminants (Fig. 517). The body is ovoid in shape, while the anterior end has a spiral row of cirri leading

to a cytostome and œsophagus. The posterior end of the body is prolonged into several caudal processes. There is an elongate macronucleus extending along one side of the body, and a small micronucleus. One or more contractile vacuoles are present.

Genus: **Cunhaia Hasselmann, 1918.**

This genus, which is very closely allied to *Entodinium*, was founded by Hasselmann (1918) for a parasite of the cæcum of the wild guinea-pig, *Cavia aperea*, of Brazil (Fig. 518). The single species of this genus,
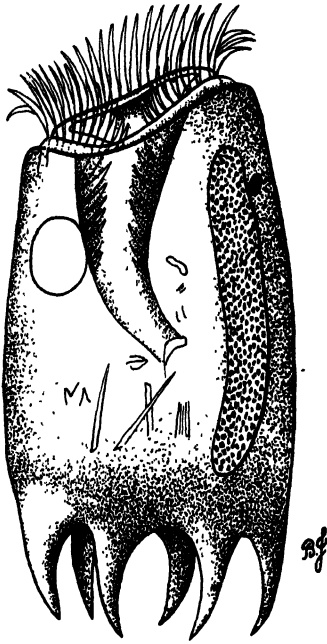
FIG. 517.—*Entodinium dentatum* FROM STOMACH OF CATTLE (× 1,000). (AFTER EBERLEIN, 1895.)

FIG. 518.—*Cunhaia curvata* FROM THE CÆCUM OF WILD GUINEA-PIG (*Cavia aperea*) BRAZIL (× *ca.* 1,000). (AFTER HASSELMANN, 1925.)

C. curvata, has an elongate curved cylindrical body, the convex surface being dorsal and the concave ventral. There is a spiral row of cirri at the anterior end, as in *Entodinium*, but a zone of cilia, absent in *Entodinium*, extends along the anterior third of the dorsal surface of the body. There is a long macronucleus in the dorsal region, and near its centre is the micronucleus. There is an anterior and a posterior contractile vacuole near the dorsal surface. The ciliate measures 60 to 80 microns in length by 30 to 40 in breadth.

Genus : **Lavierella** Buisson, 1923.

This genus was founded by Buisson (1923*a*) for a ciliate discovered by him in the intestine of the rhinoceros (Fig. 519). There is a single species, *Lavierella africana.* It bears some resemblance to members of the genus *Entodinium*, but differs in the structure of the buccal apparatus and in the possession of a spherical macronucleus. The ciliate measures 45 to 55 by 20 to 25 microns. A cytostome is present at the centre of the anterior end, which is uniformly covered with long cilia and separated from the rest of the body by a deep constriction. At the posterior end is a notch
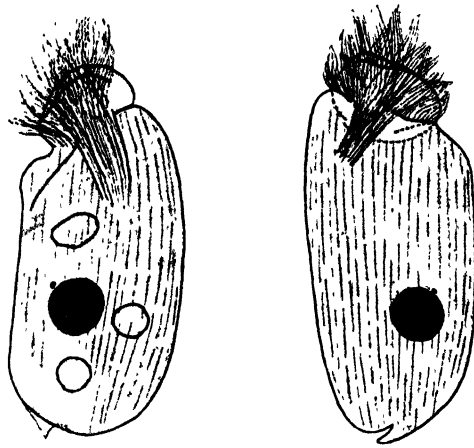
FIG. 519.—*Lavierella africana* FROM INTESTINE OF RHINOCEROS (× 1,000). (AFTER BUISSON, 1923.)

in which the anal aperture is found. At the centre of the body or a short distance behind this point lies the spherical macronucleus, 5 to 8 microns in diameter. Near it is found the micronucleus.

Genus : **Diplodinium** Schüberg, 1888.

There are several species of this genus, which was established by Schuberg (1888). The anatomy of *D. ecaudatum* has been studied by Sharp (1914). The body is elongate, and at one side of the blunt anterior end there is a peristome region with a spiral row of cirri (Fig. 520). At the opposite side of the anterior end of the body is a second spiral of cirri, which surround a pit and distinguish this genus from the preceding ones. The posterior end of the body is either rounded or provided with caudal processes. The internal structure as also that of the peristome region, as described by Sharp, is exceedingly complicated. A long macronucleus

extends along one side of the body, and at its centre is the micronucleus. There is an anterior and a posterior contractile vacuole, while an anal
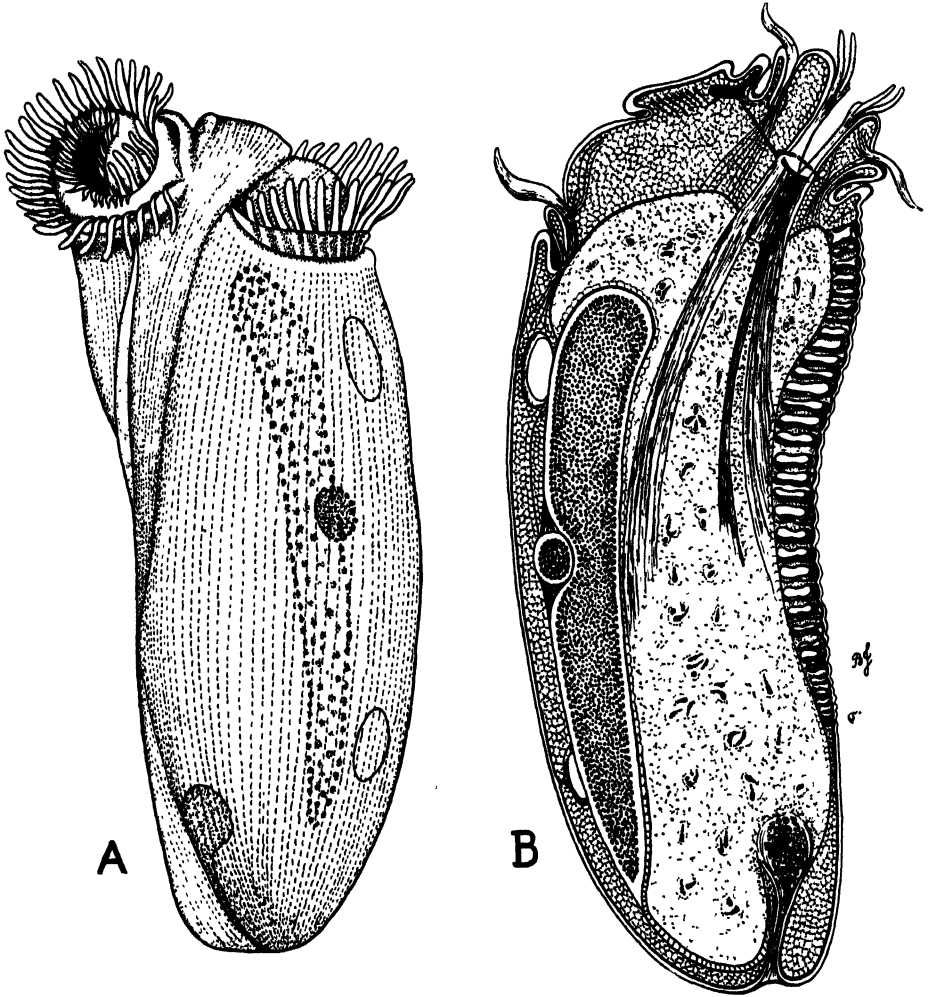
FIG. 520.—*Diplodinium ecaudatum* FROM THE STOMACH OF CATTLE (× *ca.* 1,000). (AFTER SHARP, 1914.)

1. View of left side of body, showing macronucleus, micronucleus, contractile vacuoles, and other structures.
2. Median sagittal section, showing the structure of the ectoplasm and endoplasm and the various fibrils which bring about retraction of the mouth region. The openings of the contractile vacuoles and the anus are shown.

aperture is present at the posterior end. The various species of *Ento-dinium*, which differ from one another chiefly in the character of the posterior end of the body, are parasites of the stomach of ruminants.

According to Awerinzew and Mutafowa (1914), the second spiral of cirri mentioned above is at one end actually continuous with the adoral zone of cirri.   In certain forms, however, there is a complete break between the two, the adoral zone being quite separate from the second spiral of
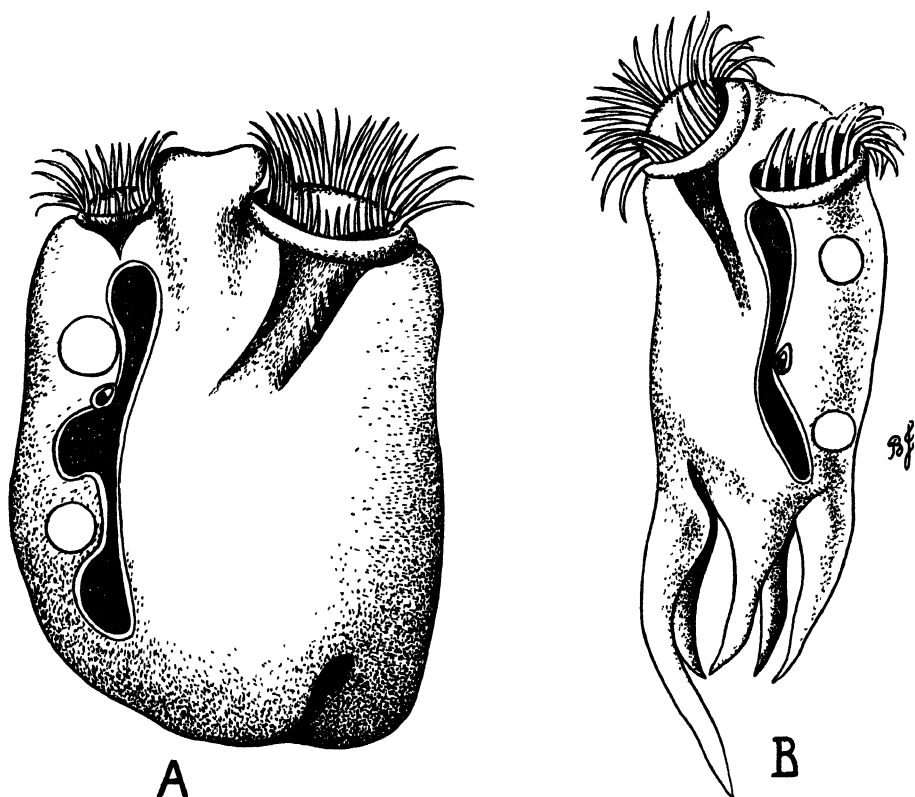
FIG. 521.—CILIATES FROM STOMACH OF CATTLE.   (AFTER AWERINZEW AND MUTAFOWA, 1914.)

A. *Metadinium medium* (× ca. 400).   B. *Ophryoscolex fasciculus* (× ca. 600).

cirri.   These forms have been placed by them in a new genus *Metadinium*, there being a single species, *M. medium*, from the rumen of cattle (Fig. 521, A).

Crawley (1924) points out that in other forms the second spiral of cirri is replaced by a band of cirri on the dorsal surface a short distance from the anterior end.   This band encircles about one-half the girth of the body.   For these forms he establishes the new genus *Epidinium*.

Genus: **Spirodinium Fiorentini, 1890.**

This genus was founded by Fiorentini for a ciliate which he named *Spirodinium equi* (Fig. 522). It measures 230 by 60 microns, and has a spiral band of cilia round the body and an elongate macronucleus. It occurs in the cæcum of the horse.

Genus: **Triadinium Fiorentini, 1890.**

There is one species, named by Fiorentini *Triadinium caudatum* (Fig. 522, 2). The ciliate, which was found in the cæcum of a horse, measures 130 by 90 microns. It has three circlets of cilia on the body and a posterior caudal tuft.
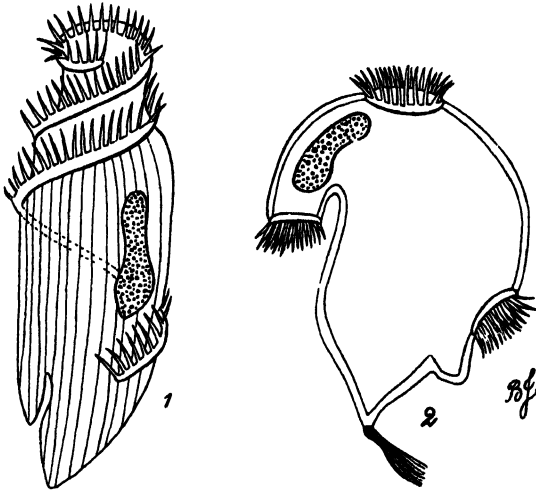
FIG. 522.—CILIATES FROM THE CALCIUM OF THE HORSE.

1. *Spirodinium equi* (× ca. 30).
2. *Triadinium caudatum* (× ca. 20).

(FROM BUISSON, 1923, AFTER GEDOELST.)
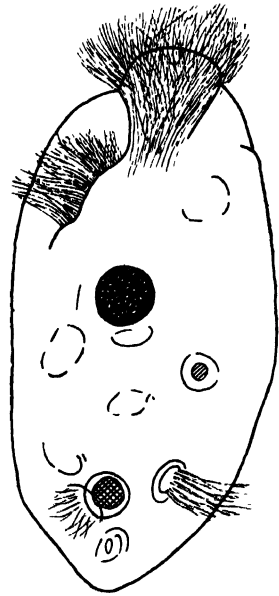
FIG. 523.—*Bozasella rhinocerotis* FROM THE INTESTINE OF THE RHINOCEROS (× 1,000). (AFTER BUISSON, 1923.)

Genus: **Bozasella Buisson, 1923.**

This genus, of which there is but one species, *Bozasella rhinocerotis*, possesses some of the characters of *Lavierella* and *Cycloposthium* (Fig. 523). The body of *B. rhinocerotis*, which measures 60 to 75 by 30 to 40 microns, is ovoid and has an anterior ciliated region separated by a constriction. Posteriorly there are two ciliated processes (caudalia) close together, while on the anterior part of the opposite surface of the

body occurs an oblique line of long cilia. The nucleus is spherical and centrally placed. It measures 8 to 10 microns in diameter. Neither the cytostome nor the anal aperture has been detected in the few specimens available for examination. The ciliate is a rare parasite of the intestine of the rhinoceros.

Genus: **Ophryoscolex** Stein, 1858.

Stein established this genus for certain ciliates which he noted in the stomach of cattle. He named two species, while Eberlein (1895) described a third from the stomach of sheep. The body of a typical member of the genus is pyriform in shape, and has at its anterior end a spiral adoral zone of cirri which pass through the cytostome into the œsophagus. Near the junction of the anterior and middle thirds of the body is an incomplete circle of cirri, while at the posterior end is a number of caudal processes. An elongate macronucleus occupies one side of the body, and at its centre is a micronucleus. There are several contractile vacuoles (Fig. 521, B).

Genus: **Cycloposthium** Bundle, 1895.

This genus was founded by Bundle (1895) for a ciliate named by him *Cycloposthium bipalmatum*, which occurs in the cæcum of the horse (Fig. 524, A). The body is barrel-shaped and has at its anterior end a circle of cirri surrounding a cone-shaped elevation, at the top of which is the cytostome. On each side of the posterior end of the body there projects a caudal appendage which is divided peripherally into a number of tapering processes. There is a long macronucleus on one side of the body, and near its centre is the micronucleus. A row of contractile vacuoles occurs beside the macronucleus.

Da Cunha (1915) has described three species of *Cycloposthium* from the cæcum of the capibara (*Hydrochœrus capybara*) of Brazil.

Genus: **Prototapirella** Cunha, 1918.

This genus was founded by da Cunha (1918) for a ciliate which in many respects resembles the species of *Cycloposthium*. In addition to the two caudal appendages, however, there are two others on that side of the body on which the macronucleus lies (Fig. 524, B). One is situated near the middle of the body, and the other near the anterior end. The ciliate was found in association with others in the stomach of tapirs (*Tapirus americanus*) in Brazil. It measures from 80 to 140 microns in length by 60 to 120 microns in breadth.

Buisson (1923) has given the names *Prototapirella cristata* and *P. clypeata* to two forms in the rhinoceros, and the name *P. elephantis* to one in the elephant.

Genus : **Tripalmaria** Gassowsky, 1919.

The single species of this genus, *Tripalmaria dogieli*, resembles members of the genus *Prototapirella*, except in the number of appendages,
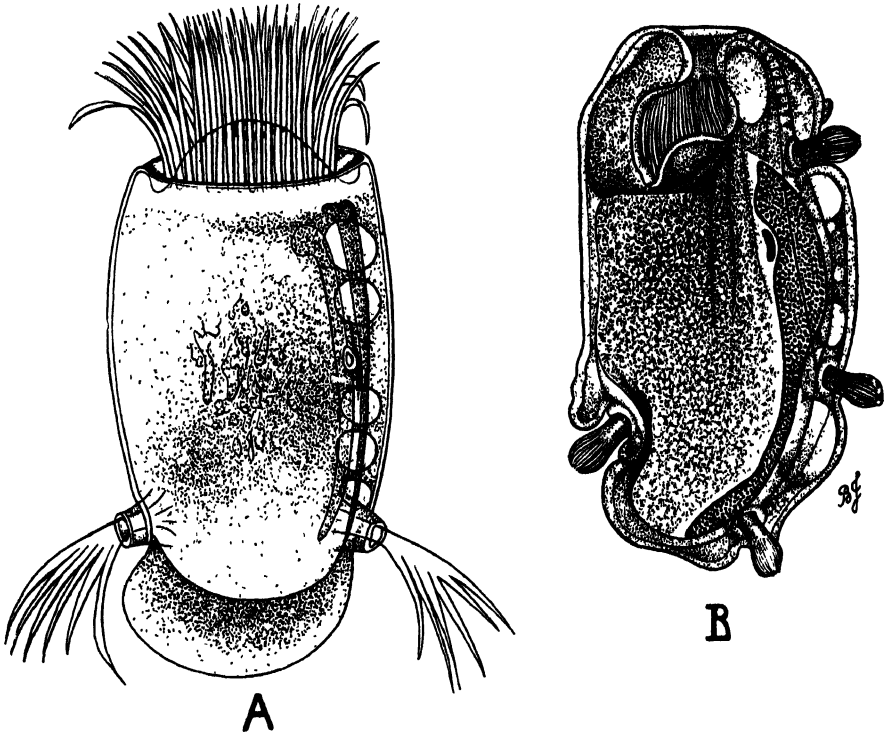
FIG. 524.—CILIATES FROM THE CÆCUM OF THE HORSE AND TAPIR. (A, AFTER BUNDLE, 1895; B, AFTER DA CUNHA, 1919.)

A. *Cycloposthium bipalmatum* from the horse (× *ca.* 450).
B. *Prototapirella intestinalis* from the tapir; anterior region retracted (× *ca.* 700).

of which there are two at the posterior end of the body and one, dorsal in position, near the anterior end (Fig. 525, 1). Around the cytostome is a circlet of membranes. The macronucleus consists of two irregular lobes, near the ventral of which is the micronucleus. The ectoplasm on the right side of the body forms a supporting structure, the margins of which are thickened to form two longitudinal ridges. The ciliate, which lives in the colon of the horse, measures 99 to 210 by 55 to 91 microns.

Genus : Tricaudalia Buisson, 1923.

This genus was founded by Buisson (1923) for a ciliate named by him *Tricaudalia brumpti,* which he discovered in the rhinoceros. It resembles *Tripalmaria dogieli* in the possession of only three appendages. It is possible that it actually belongs to the same genus.

Genus : Tetratoxum Gassowsky, 1919.

There is a single species, *Tetratoxum unifasciculatum,* which was placed by Fiorentini (1890) in the genus *Diplodinium,* and by Sharp (1914) in the genus *Blepharocorys* (Fig. 525, 2). It was made the type of a new genus by Gassowsky (1919). The body is not covered with cilia, but is provided
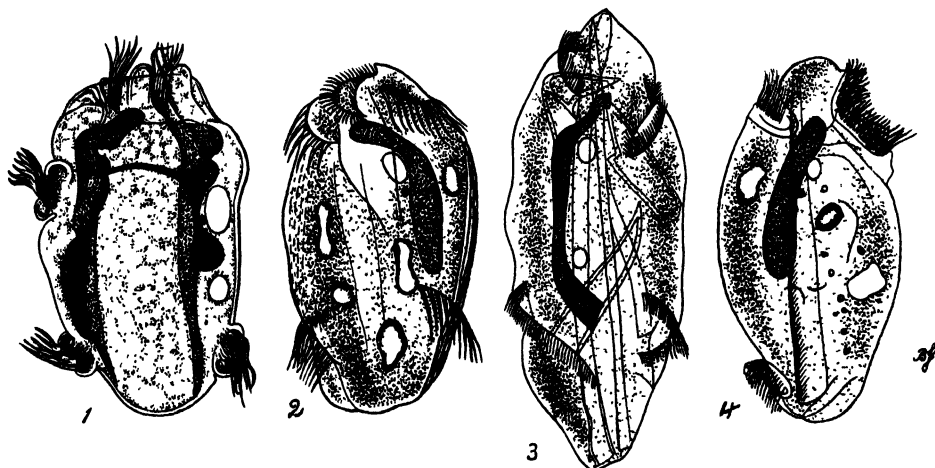
FIG. 525.—CILIATES FROM THE CÆCUM OF THE HORSE.   (AFTER GASSOWSKY, 1919.)

1. *Tripalmaria dogieli* (× 270).   2. *Tetratoxum unifasciculatum* (× 270).
3. *Cochliatoxum periachtum* (× 130).   4. *Ditoxum funinucleum* (× 270).

with arches composed of a series of membranes. On the anterior end of the body are two such arches, and two others occur on the posterior end. At the anterior end is a slit-like cytostome surrounded by minute cilia. The body, which is somewhat flattened, has dorsal and ventral surfaces, on each of which are six to eight ridges or ribs. In the hollow of the macronucleus is the contractile vacuole. The ciliate, which occurs in the colon of the horse, measures 91 to 179 by 49 to 81 microns.

Genus : Cochliatoxum Gassowsky, 1919.

There is a single species, *Cochliatoxum periachtum,* which resembles *Tetratoxum unifasciculatum* in many respects (Fig. 525, 3). It is also an inhabitant of the colon of the horse, but is larger, and measures 400 to

510 by 215 to 235 microns. There are two arches of membranes at either end of the body, and two contractile vacuoles in the concavity of the macronucleus.

Genus: Ditoxum Gassowsky, 1919.

The single species of this genus, *Ditoxum funinucleum*, resembles the two preceding forms in most respects, but differs in that there is only a single posterior arch of membranes (Fig. 525, 4). It is found in the colon of the horse, and measures 145 to 225 by 72 to 108 microns.

Genus: Troglodytella Brumpt and Joyeux, 1912.

This genus was founded by Brumpt and Joyeux (1912) for a curious ciliate seen in the fæces of a chimpanzee, and to which they gave the name

FIG. 526. — *Troglodytella abrassarti* FROM LARGE INTESTINE OF CHIM-PANZEE (× 270). (AFTER REICHE-NOW, 1920.)

Troglodytella abrassarti. This organism was studied in the Cameroons by Reichenow (1920*a*), who came to the conclusion that there were two varieties, one with a pointed posterior end, *T. abrassarti acuminata*, and the other with a rounded posterior end, *T. abrassarti abrassarti*, the one seen by Brumpt and Joyeux. Reichenow also described as *T. gorillæ* another form from the gorilla. These ciliates are allied to *Entodinium* and *Diplodinium*. The body is roughly egg-shaped, and there is a circlet of cirri at the anterior end round the cytostome (Fig. 526). In addition, there are several bands of cirri round the body. These are arranged in incomplete circlets, and give the impression that if they were continuous they would form a spiral of three turns. The breaks in this spiral produce several bands of cirri, which are arranged differently in the ciliates of the gorilla and chimpanzee owing to the intervals occurring at different places. The anterior third of the body is covered by a peculiarly marked cap, which appears to be a thickening of the periplast. On each side of the body the cap is continued posteriorly as a lappet, which appears to produce the breaks in the spiral arrangement of the cirri. The macro-nucleus is an elongate structure lying near the dorsal surface, and at its

centre is a micronucleus. There is a number of contractile vacuoles, which are arranged parallel to the bands of cirri on the left side of the body, while an anal aperture is present on the right side at a point corresponding with the junction of the middle and posterior thirds of the body. These ciliates of the chimpanzee and gorilla resemble one another so closely that it would seem reasonable to regard the differences which Reichenow describes as being merely individual variations in single species.

The parasite first seen by Brumpt and Joyeux (1912) was in a sick chimpanzee, but Reichenow found that healthy animals are also usually infected.

(3). *Order :* HYPOTRICHIDA DELAGE AND HÉROUARD, 1896.

The ciliates belonging to this order (=Hypotricha Stein, 1859) are distinctly flattened dorso-ventrally, and in association with a creeping
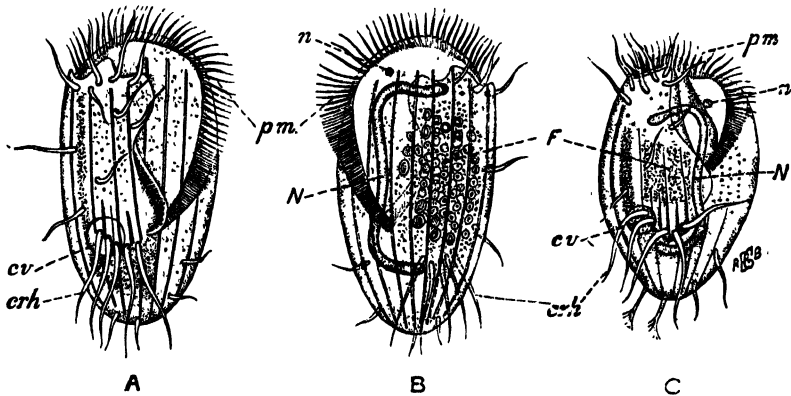
FIG. 527.—*Euplotes patella* AND *E. harpa* (×ca. 240). (FROM MINCHIN, 1912, AFTER STEIN.)

A, Ventral view of *E. patella ; B,* dorsal view of *E. patella ; C, E. harpa ; N,* macronucleus; *c.v.,* contractile vacuole; *crh,* cirri; *p.m.,* peristome membrane; *F,* food vacuole area.

mode of existence there is a special development of the cilia on the ventral surface of the body. Many of these become fused in groups to form stout processes, known as cirri, which enable the organism to rest upon and by their movements to creep over any surface. No definitely parasitic forms are known, but the ciliates are sometimes seen upon the surface of aquatic invertebrates. Two of the best-known genera are *Stylonychia* and *Euplotes.* *Stylonychia mytilus* varies in length from 100 to 300 microns, and is about three times as long as it is broad (Fig. 9). There is a flattened ventral surface, on the left-hand side of the anterior part of which is the peristome with its adoral zone of cilia. The margins of the body are provided with cirri, while groups of stouter cirri are distributed over the ventral surface. The ovoid macronucleus with the closely applied

micronucleus lies immediately behind the cytostome. The dorsal surface, which is convex, is provided with longitudinal rows of cilia. This organism is a very common inhabitant of water containing algæ or other vegetation. The members of the genus *Euplotes* are very similar to species of *Stylonychia*. They differ, however, in that the marginal cilia are reduced or absent, while there is a greater development of the posterior cirri (Fig. 527). They are usually shorter in proportion to their breadth than members of the genus *Stylonychia*.
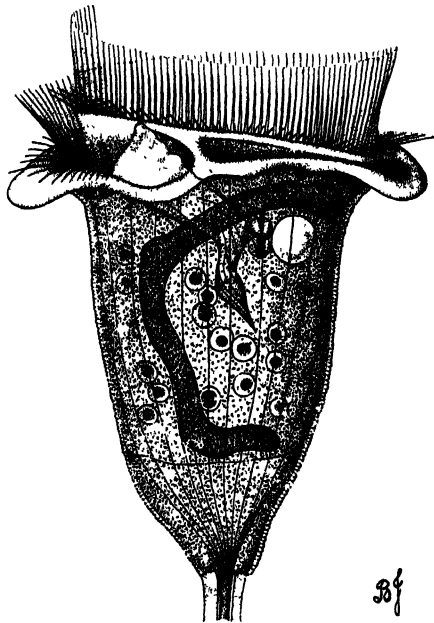
FIG. 528.—*Carchesium polypinum* (× ca. 1,000). (AFTER DOFLEIN, 1916.)

There are two spirally arranged rows of cilia; one row, consisting of short cilia, extends as far as the opening of the vestibulum. The other row has long cilia, which extend into the cytopharynx, and are fused into a membrane except at a point near the opening of the vestibulum. There is a large horseshoe nucleus, small micronucleus, and a contractile vacuole, which discharges into a reservoir between it and the cytopharynx. The cytoplasm contains numerous food vacuoles. The ectoplasm shows longitudinal fibrils, which extend into the central contractile part of the peduncle.

(4). *Order :* PERITRICHIDA DELAGE AND HÉROUARD, 1896.

In this order (=Peritricha Stein, 1859) are included ciliates which have adoral cilia in the form of a right-handed spiral. As a rule no other cilia are present, and the body is more or less cone-shaped, the apex of the cone being attached to objects either directly or through the intermediary of a filament which may be a simple thread capable of contraction like a spring, or a system of threads subdivided like the branches of a tree.

In some cases, however, the organisms are free-swimming, and this applies also to certain stages in the development of those forms which are attached when fully grown.

The free-swimming forms, in addition to the adoral cilia, have a circle of cilia round the posterior end of the body, and in some cases a definite suctorial apparatus. The attached forms are familiar objects in fresh water. The various species of *Vorticella* (Fig. 19) and its allies are found attached singly by contractile filaments to aquatic plants, and even insect larvæ. In the case of *Epistylis* and similar forms, numerous individuals
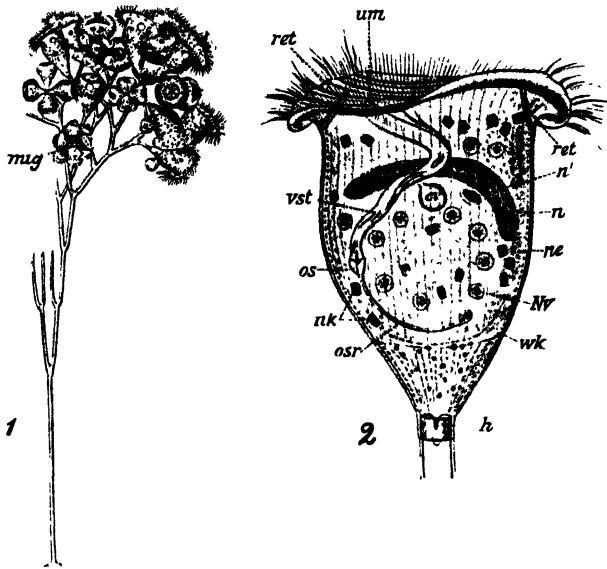
FIG. 529. —*Epistylis umbellaria*. (AFTER BÜTSCHLI, 1887-1889.)

1. Portion of colony with several clusters of microconjugants (*mig*), some of which are in conjugation with the larger macroconjugants.
2. Single individual more highly magnified (× 150). *vst*, Vestibulum; *os*, mouth; *osr*, œsophagus; *um*, undulating membrane; *n*, macronucleus; *n'*, micronucleus; *cv*, contractile vacuole; *Nv*, food vacuole; *ret*, retractile myonemes; *nk*, Nessel capsules; *wk*, ring left when posterior circlet of cilia of free-swimming form disappear; *h*, band at union of peduncle with body; *pe*, pellicle.

are united on a complicated system of branching filaments with a common stem (Fig. 529). Some of the attached forms have no filament, but are provided with chitinous cup-like shells fixed to various objects. Through the opening of the cup the ciliate can protrude its ciliated anterior end for the purpose of capturing food, and it can quickly withdraw when stimulated. Attached to the side of the body near the anterior end there may be a round operculum which closes the opening of the shell when the ciliate retracts. There are numerous free-swimming forms, unprovided with filament, which are mostly ectoparasites of fish, amphibia,

molluscs, worms, echinoderms, and other aquatic creatures. Such forms are *Spirochona*, found on the gills of the fresh-water crustacean, *Gammarus pulex; Licnophora*, which is an ectoparasite of various marine animals; and *Trichodina*, seen commonly on the skin or in the bladder of fresh-water vertebrates, such as fish and newts (Fig. 530).

The ciliated region of the body of the Peritrichida, which is to be regarded as the ventral surface, corresponds with the peristome (Fig. 528). The adoral row of cilia, which in some forms consists of two parallel rows, commences at a point on the ventral surface, and follows a course like that of a flat watch-spring till its outer end passes into a cone-shaped depression, the vestibulum, within which is the cytostome leading to the œsophagus
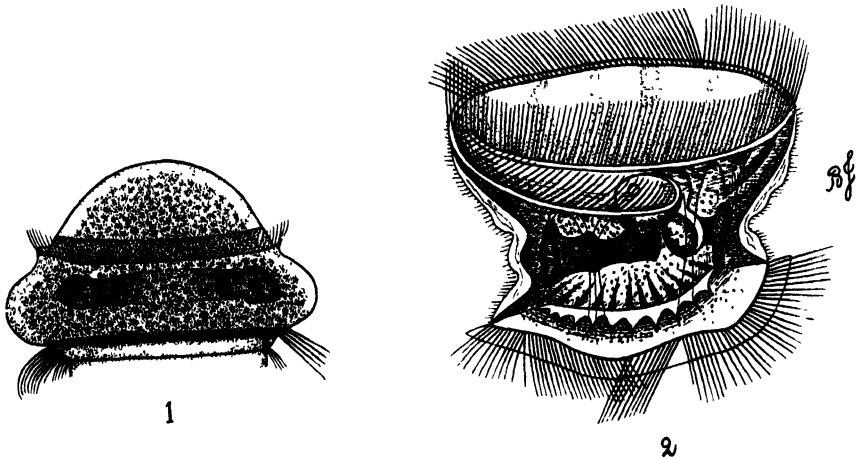
FIG. 530.—SPECIES OF *Trichodina* (× 1,000). (1, AFTER CHATTON, 1910; 2, AFTER BUTSCHLI, 1882.)

1. *T. labrorum* from the skin of marine fish of the genus *Symphodus*.
2. *T. pediculus* from the skin of fresh-water fish or the bladder of newts.

(Figs. 528, 529). The cilia may be continued into the œsophagus as cilia, or they may fuse to form an undulating membrane. The anal aperture, through which the residue from digested food is discharged, occurs in the vestibulum, as also the opening through which the contractile vacuole empties itself. Reproduction takes place by budding or binary fission. In the latter case, an example of which is seen in *Epistylis*, two equal-sized individuals arise and remain attached to a common stalk. Each forms a new stalk for itself, so that a branching system results. In the budding process which occurs in *Vorticella* and other forms there is separated a small individual provided with a ring of cilia round its posterior end. After leading a free-living existence for some time, it attaches

itself, develops a filament, and grows into the adult type, or it may conjugate with a larger individual, during which the nuclei undergo complicated changes which have been described above (Fig. 44).

VII. *CLASS:* SUCTORIA Claparède and Lachmann, 1858.

The members of this class differ from the Ciliata in that cilia are present only in the young stages, which are budded off from the adults, while the adults themselves are provided with sucking tentacles. The young forms, often called embryos, lead a free-swimming exist-
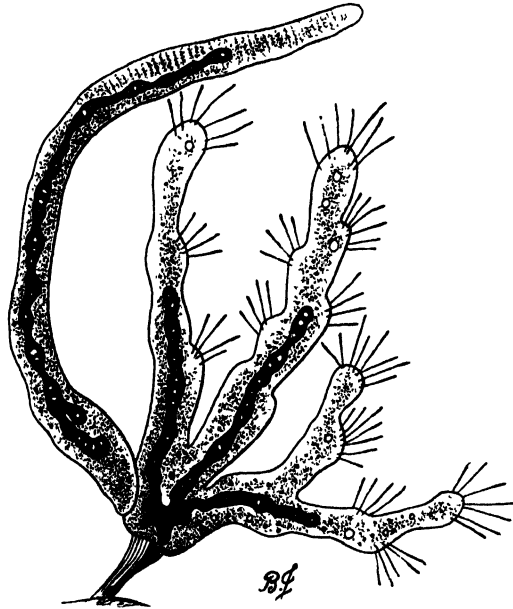
FIG. 531.—*Dendrosomides paguri:* Parasitic on Crustacea of the Genus *Eupagurus* (× 300). (After Collin, 1912.)

ence for a time. They then usually attach themselves to objects, lose their cilia, and develop suctorial tentacles, by means of which they are able to imbibe nourishment from material or organisms which become adherent to them (Fig. 15). The Suctoria are devoid of cytostome, but one or more contractile vacuoles are present. The nuclear arrangement resembles that of the Ciliata, there being both macronuclei and micronuclei. The majority of the Suctoria are in the adult stages pedunculated organisms attached to algæ, the bodies of small Crustacea, aquatic larvæ, or other objects. In some cases complicated branched systems are produced (Fig. 531). A few unattached forms have been described, and

these in the adult stages have more or less globular bodies provided with a number of radiating tentacles. One such form, *Sphærophrya pusilla*,
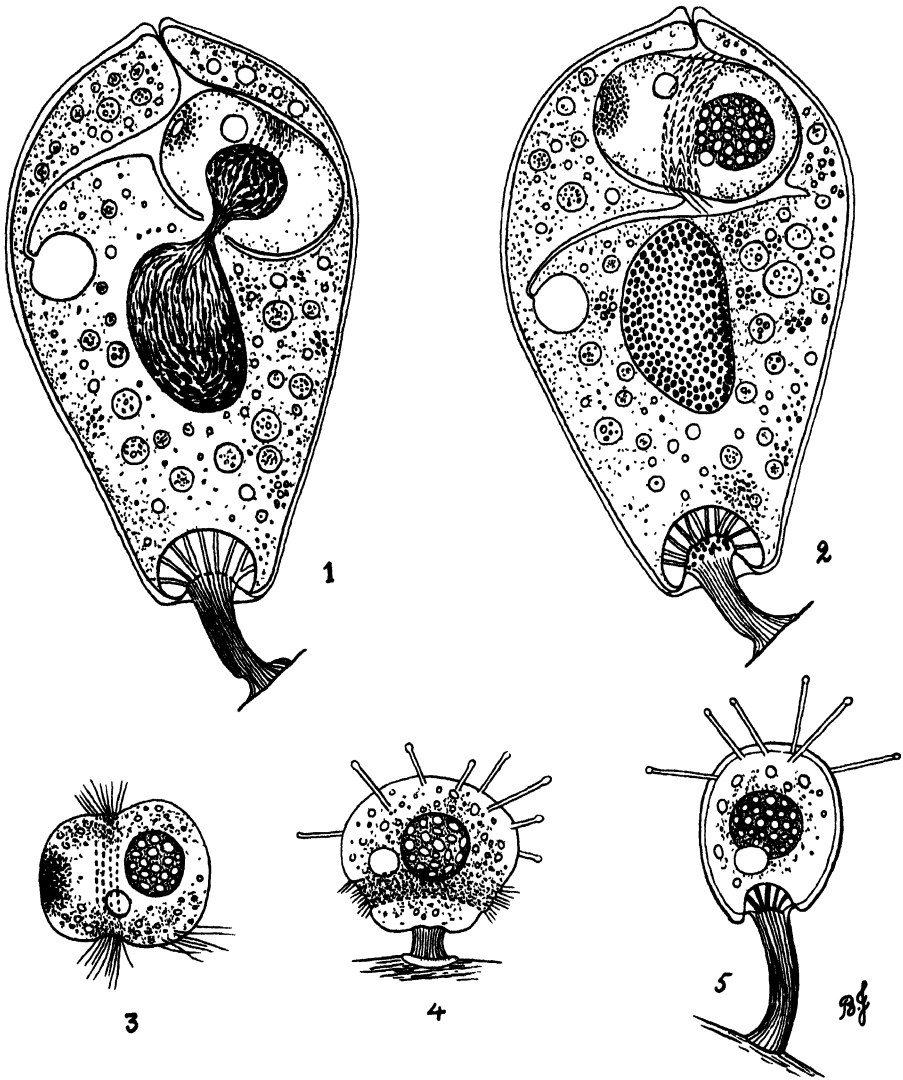
FIG. 532.—*Tokophrya cyclopum:* BUDDING PROCESS AND DEVELOPMENT OF CILIATED EMBRYO (×1,000.) (AFTER COLLIN, 1912.)

1. Formation of internal bud and division of parent nucleus.
2. Ciliated embryo in pouch of parent.   3. Free ciliated embryo.
4. Attached form with tentacles and cilia still present.
5. Later stage after disappearance of cilia.

is parasitic on free-living Ciliata, and another, *Allantosoma intestinalis*, attacks the ciliates which inhabit the colon or cæcum of horses.

Reproduction may take place by binary fission, but most usually by production of ciliated embryos, which are formed by a process of budding. There may be external budding, whereby one or more buds are found on the surface of the organism (Fig. 40), or internal budding, in which the buds are produced within a kind of brood sac which has resulted from a deep depression of the surface of the body. In this sac or cavity, which communicates with the exterior by a pore, one or more buds are formed (Fig. 532). The buds become separated within the sac and, like those
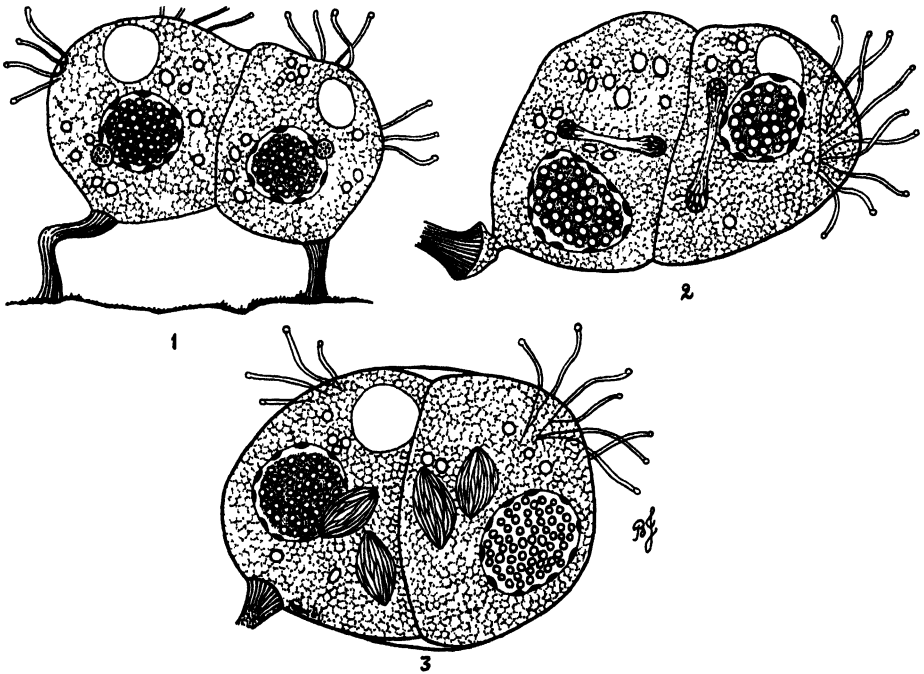
FIG. 533.—*Tokophrya cyclopum:* STAGES IN CONJUGATION (× 1,360). (AFTER COLLIN, 1912.)

1. Two associated individuals.   2. Division of micronuclei.
3. Two nuclei in each conjugant, possibly conjugating nuclei.

formed by the external budding process, develop cilia, which are usually arranged in circlets. For some time the ciliated embryo moves about within the sac, but finally it escapes through the pore and swims away. It eventually becomes attached to any suitable object, which may be the body of an aquatic larva or crustacean. It loses its cilia, develops tentacles, and grows into the adult pedunculated form. In the budding process the nuclei of the parent become divided to supply the nuclei of the ciliated embryos.

A sexual process has been observed in some forms, but the details have not been so fully worked out as in the case of many Ciliata, though enough is known to indicate that it is very similar in the two groups. Two organisms associate, after which the macronuclei degenerate, while the micronuclei undergo a number of mitotic divisions. Exchange and union of micronuclei takes place, and after separation of the organisms the macronuclei are reformed from products of division of the micronuclei (Fig. 533).

The majority of Suctoria are non-parasitic, though some of them, such as species of *Dendrosoma* and *Dendrosomides* (Fig. 531), may be found
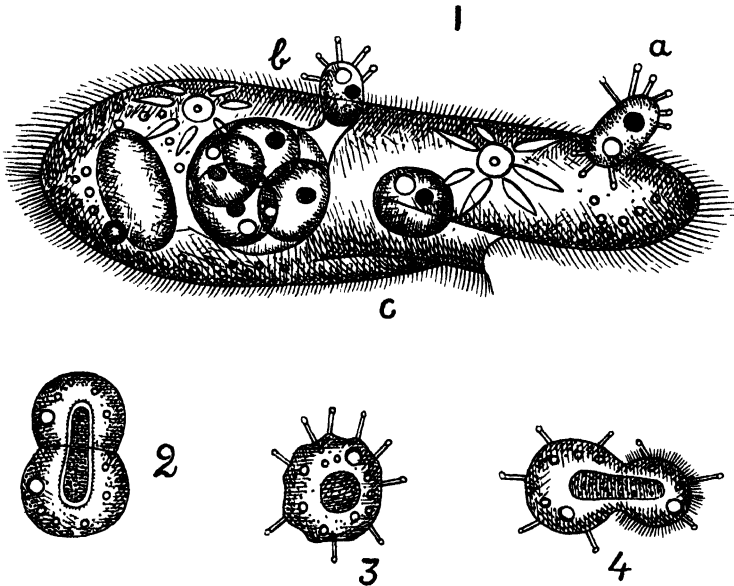
FIG. 534.—*Sphærophrya pusilla* PARASITIC IN CILIATES. (AFTER BÜTSCHLI, 1882.)

　1a. Embryo making its way into the cytoplasm of *Paramœcium caudatum* (× 500).
　1b. Vacuole containing four organisms, from the opening of which an embryo is escaping.
　1c. Single organism in the cytoplasm.
　2. Dividing form, as seen in the vacuole (× ca. 750).
　3. Free form after escape from vacuole. Cilia have not yet been developed (× ca. 750).
　4. Free-swimming embryo with tentacles and cilia (× ca. 750).

attached in enormous numbers to the gills or other parts of the bodies of fresh-water Crustacea or aquatic larvæ.

Sphærophrya pusilla Claparède and Lachmann, 1858.—This form, which reaches a diameter of about 15 microns, is parasitic on ciliates, such as species of *Paramecium, Stentor*, and *Stylonychia* (Fig. 534). The infected ciliates are seen to possess large vacuole-like spaces which communicate with the exterior by a pore. Within each vacuole is a varying number of rounded cytoplasmic bodies with single nuclei. These escape

through the pore and swim by means of cilia.   Tentacles are developed, and they attach themselves to other ciliates.   They gradually sink into the cytoplasm, being finally enclosed in a sort of vacuole which communicates with the exterior.   Within the vacuole multiplication takes place by binary fission or a budding process which gives rise to the ciliated embryos.   As many as fifty organisms may occur in a single vacuole. Maupas (1881) has recognized two other species—*Sphærophrya magna* and *S. stentoris* (Fig. 15).   They reach a diameter of 50 microns, the latter being parasitic on *Stentor roeseli*.   Another species is *Spærophrya soliformis* described by Lauterborn (1908).

Allantosoma intestinalis Gassowsky, 1919.—This suctorian lives in the colon and to a less extent in the cæcum of horses, where it attacks
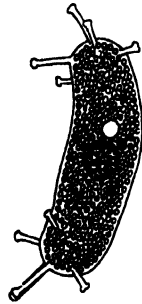
FIG. 535.—*Allantosoma intestinalis:* PARASITIC ON CILIATES IN CÆCUM OF HORSE (× 550). (AFTER GASSOWSKY, 1919.)

the ciliates which occur in the same locality (Fig. 535).   It has an elongate, sausage-shaped body, varying in length from 16 to 65 microns and in breadth from 5 to 27 microns.   Each end is provided with several suckers, by means of which the organism attaches itself to the ciliates, which it parasitizes.   There is a spherical macronucleus, near which is the micronucleus and a contractile vacuole.

PART III
SPIROCHÆTES

SPIROCHÆTES

GENERAL ACCOUNT OF STRUCTURE, AFFINITIES, AND NOMENCLATURE.

THE name Spirochæte is employed as a general term for certain spiral organisms which have flexible bodies. Some forms which are parasitic in the crystalline styles of oysters and other Mollusca are comparatively broad, so that accurate details of their structure can be obtained. The majority, however, are so slender that there has been considerable difference of opinion as to their morphology. The spiral form of the body and its flexibility are not in themselves characteristic of the group, for there occur amongst the bacteria similar spiral organisms of the genus *Spirillum*, which have rigid bodies, while definitely flexible bacteria are also known. Certain Cyanophyceæ, or coloured algæ, such as *Spirulina*, which consists of a single chamber, and *Arthrospira*, which has transverse septa, likewise have spiral bodies (Fig. 536). In consequence of this, some authorities have classed the spirochætes with the bacteria, and others with the algæ. According to Dobell (1911), the minute structure of the larger spirochætes is quite different from that of the bacteria and the Cyanophyceæ, and he concludes that the spirochætes form a special group of the Protista, which he calls the Spirochætoidea. Fantham (1908) had already proposed the name Spirochætacea and Gross (1910) the name Spironemacea for essentially the same group of organisms.

At the present time it is difficult to account for Schaudinn's view that spirochætes are Protozoa, for they have no definitely constituted nuclei, reproduce by transverse fission, and are not orientated into an anterior and posterior end. The limiting membrane of the body is quite different in structure from that of the flagellates with which he associated them. It is evidently impossible to regard the spirochætes as Protozoa; they appear to be much more closely allied to the bacteria, and in this respect may be regarded as nearer to plants than to animals. The fact that certain blood-inhabiting spirochætes are transmitted by lice and ticks from one host to another has been advanced as an argument in favour of their Protozoal nature, and attempts have been made to trace a cycle of development in these arthropods which can be compared with the invertebrate cycles of trypanosomes. It is known, however, that *Bacillus pestis* of plague is transmitted by fleas, and that certain undoubted unicellular vegetable organisms have complicated life-histories in which a process of syngamy occurs. It is, therefore, quite illogical

to assume that, because an organism is conveyed by a biting arthropod or has a complicated life-history, it must necessarily be a Protozoon. Schaudinn's supposition that *Leucocytozoon ziemanni* gave rise to innumerable spirochætes in the stomach of the mosquito has been found to be entirely erroneous (see p. 904).

As at present understood, the group Spironemacea or Spirochætoidea includes organisms of several distinct types (Fig. 537). The name *Spirochæta* was first given by Ehrenberg (1834) to a large, flexible spiral organism which occurs in water. The type species *Spirochæta plicatilis* Ehrenberg, 1834, may be as much as 500 microns in length and 0·5 to 0·75 micron in breadth. According to Zuelzer (1910), the spiral body is strengthened by an axial rod or filament, so that the entire organism has very much the appearance of a piece of rubber tubing wound round a piece of fine wire, the wire representing the axial rod. Organisms which have this structure are included in the genus *Spirochæta*. Another type of organism is characterized by the possession of a membranous structure which was called the crista by Gross (1910). Such a form is *Cristispira balbianii*, which occurs commonly in the crystalline style in the stomach of oysters (Fig. 540). If the axial rod of the members of the genus *Spirochæta* be regarded as broadened into a membrane, the characteristic arrangement of *C. balbianii* would result (Fig. 537).

FIG. 536.—*Spirulina versicolor* (× *ca*. 500). (AFTER ZUELZER, 1911.)

1. Appearance of living organism.
2. Specimen fixed and stained, showing granules.

These two genera, which include comparatively large organisms, appear to be well defined, but there is a number of much smaller forms, including the various pathogenic spirochætes, which are very difficult to study on account of their narrowness. The only absolutely reliable information regarding their structure is that they possess flexible spiral bodies, and it is largely a matter of conjecture that they are regarded as related to the two genera, *Spirochæta* and *Cristispira*. The accounts which have been given of their minute structure differ widely from one another. Some observers have considered that they possess crista or membranes; others that they have terminal flagella, and reproduce by both longitudinal and transverse division, and even produce spores. There is so much discrepancy in these accounts that it is evident many of the statements are unreliable. Attempts have, nevertheless, been made to subdivide the small spirochætes into a number of genera, with the result that they have been classed as *Spironema*, *Treponema*, and *Leptospira*.

The name *Spironema* was first employed by Vuillemin (1905) for the organism of syphilis, which Schaudinn had called *Spirochæta pallida*. As the name *Spironema* had been employed by Klebs (1892) for a flagellate, and as Schaudinn regarded the spirochætes as Protozoa, he (1905) proposed the name *Treponema*, and designated the spirochæte of syphilis *Treponema pallidum* (Fig. 551). It is perfectly clear that the spirochætes are not Protozoa, so that there would be no reason why Vuillemin's name *Spironema* should not be employed if it had not also been used previously for a vegetable organism. Except as regards its size and the number of turns, the spirochæte of syphilis does not differ from those which produce relapsing fever in man, and the various forms which occur in the alimentary tract or chronic ulcerations of the skin, and many other
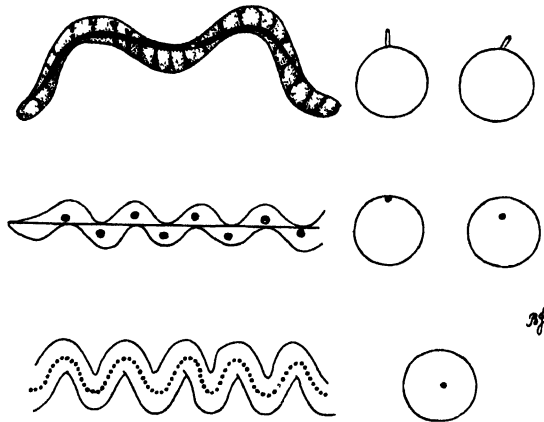
Fig. 537.—Diagram of Structure of Various Spiral Organisms as seen in Longitudinal and Transverse Sections. (After Zuelzer, 1911.)

Cristispira, showing septate condition of body and crista.
Spirochæta, showing axial filament and granules. The transverse sections show the position of the filament at different parts of the body. *Spirulina*, showing central row of granules.

situations, such as stagnant water (Fig. 542). It is better to refer all these forms to the genus *Treponema*. There still remains the genus *Leptospira*, which was established by Noguchi (1917) for the causative organism of Weil's disease, and which is now generally known as *Leptospira icterohæmorrhagiæ* (Inada and Ido, 1914). This spirochæte may be sufficiently distinct to justify its inclusion in a separate genus, but here again opinions differ. The body consists of a spirally wound thread, the spirals being so fine and close together that, unless careful inspection is made, they are overlooked. The body is exceedingly flexible, and has a very characteristic crook or hook at each end (Fig. 557). Zuelzer (1918 and 1921), whose extensive studies entitles her to speak with authority, believes that all the spiro·

chætes which are referred here to the genus *Treponema* have essentially the same structure as *Spirochæta plicatilis*, and that they possess similar axial filaments, which can be demonstrated, in some cases at least, by special technique. This structure she considers as present also in the members of the genus *Leptospira*. On this account, she proposes to return all the various spirochætes which are included in the genera *Treponema* and *Leptospira* to the genus *Spirochæta*.

The difficulty associated with the nomenclature is well illustrated by an organism which was named *Spirochæta regaudi* by Ball and Roquet (1911), and which was discovered in the cells of the peptic glands of the stomach of the dog by Bizzozero (1893). It was studied by Duboscq and Lebailly (1912a), who found it in the dog, cat, and fox. They describe the organism as having three to twenty turns, a flagellum 3 to 4 microns in length at each end, and a body which is flexible like that of a spirochæte. It appeared to them to occupy a position between spirochætes and spirilla, so they established for it the new genus *Spirella*, the organism being *Spirella regaudi*. It was investigated again by Kasai and Kobayashi (1919). These observers found it in dogs, cats, wild rats, and monkeys. White rats were easily infected by oral administration of infected material. Though the Japanese observers believe the body to be rigid, Edkins (1923), who found the organism in the cells of the fundus as also the pyloric end of the cat's stomach, described it as a fixed, but flexible spiral. Whatever movements are exhibited appear to be the direct result of the action of the flagella. Another genus is *Cristispirella*, created by Hollande (1921) for an organism said to possess a definite membrane, which he found in the small intestine of the guinea-pig.

The observation of Duboscq and Lebailly noted above raises the question of the relationship of the flexible spirochætes to the rigid spirilla. It is possible that these two types are more closely related than has hitherto been supposed. Such a form as the parasite of rat-bite fever, to be considered below, behaves in many respects like a spirochæte, but it has a rigid body and terminal flagella, and is regarded here as a *Spirillum*, though Sangiorgi (1922) places it as a new genus, *Treponemella* (Fig. 562).

Mesnil (1921), in a review of the subject, states that if members of the genus *Cristispira* are excluded, he has been quite unable to discover any features which can be regarded as of sufficient generic value to justify the division of the group into separate genera. He accordingly agrees with Zuelzer that only two genera can be recognized, *Spirochæta* and *Cristispira*. The adoption of this classification would undoubtedly simplify the nomenclature, but in view of the fact that observers are by no means agreed as to the presence of an axial filament in the smaller

organisms, it appears safer at present to retain at least four genera, as follows:

1. *Genus*: **Cristispira** Gross, 1910; type species *Cristispira balbianii* (Certes, 1882).

2. *Genus*: **Spirochæta** Ehrenberg, 1834; type species *Spirochæta plicatilis* Ehrenberg, 1834.

3. *Genus*: **Treponema** Schaudinn, 1905; type species *Treponema pallidum* Schaudinn, 1905.

4. *Genus*: **Leptospira** Noguchi, 1917; type species *Leptospira icterohæmorrhagiæ* (Inada and Ido, 1914).

FREE-LIVING SPIROCHÆTES.

It has already been mentioned that spirochætes are commonly present as free-living organisms in water. The type species is the large *Spirochæta plicatilis* of Ehrenberg (Fig. 539). Many smaller forms occur, and Zuelzer (1921), Uhlenhuth and Zuelzer (1921), and others, have shown that all the various types which have been described as living either parasitically or saprophytically in man and animals have their representatives in fresh water. Attention was drawn by these observers to a free-living form which is indistinguishable from *Leptospira icterohæmorrhagiæ*. They call the organism *Spirochæta pseudo-icterogenes*, and note its resemblance to a form seen in water by Wolbach and Binger (1914), and which was named *S. biflexa*. These fresh-water spirochætes can often be found in large numbers in the mucoid material which collects round the orifice of laboratory taps. What relation they have to the parasitic or saprophytic forms is not known, but it would seem reasonable to suppose that many of the free-living types have become adapted to a saprophytic or parasitic life in animal hosts. Zuelzer (1922) describes the cultivation of the *Leptospira* of water. One strain, after it had been sub-cultured a number of times, proved to be virulent to guinea-pigs, in which it produced an infection resembling that resulting from inoculation with the organism of Weil's disease. Buchanan (1924) has also infected guinea-pigs with a leptospira occurring in the slime on the roof of a mine. In a later paper, Zuelzer and Oba (1923) describe various saprophytic spirochætes which occur in water and in the alimentary tract of man and animals. A study of these organisms reveals the fact that though forms of the relapsing fever, syphilis, and Weil's disease types occur, there are many intermediate forms which make it impossible to define accurately the genera *Treponema* and *Leptospira*. The conclusion is again reached that all these spiral organisms should be placed in the single genus *Spirochæta*. The authors prove that in pure culture the morphology of a particular species varies considerably according to the type of medium

used. An organism may resemble a typical leptospira in one medium, while in another it is indistinguishable from the organism of syphilis.

Hindle (1925) has shown that if a portion of human fæces about the size of a pea be emulsified in about 20 c.c. of London tap water, the mixture, if incubated at 25° to 30° C. for ten days in a Petri dish, will yield an abundant growth of leptospira. The organisms are most numerous on about the twentieth day, and survive for four or five weeks. That the leptospira comes from the water is proved by the fact that the same
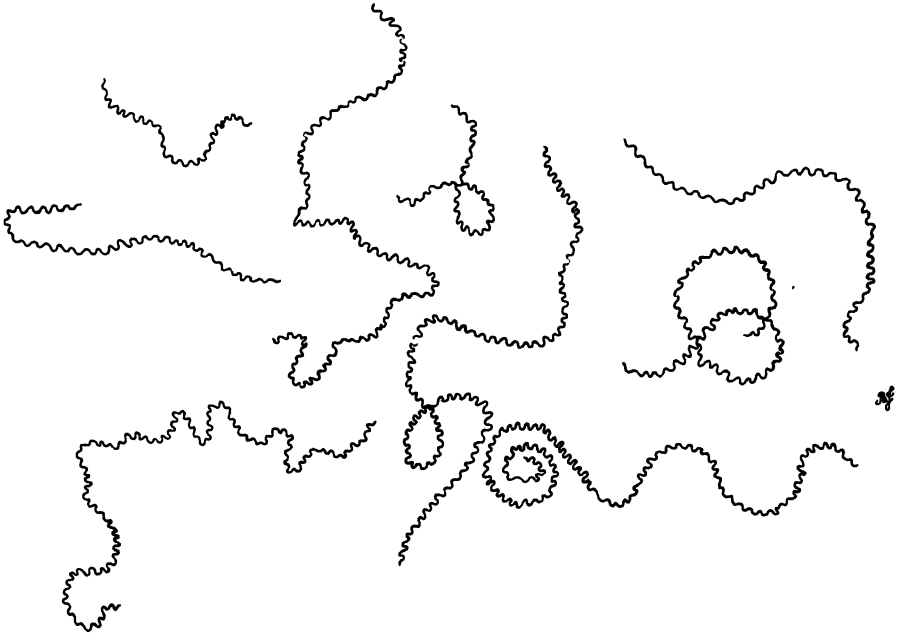
FIG. 538.—*Spirochæta plicatilis*, AS SEEN IN THE LIVING CONDITION IN WATER (× *ca.* 500). (AFTER ZUELZER, 1911.)

growth occurs if the portion of fæces is emulsified in a small quantity of water and sterilized by boiling before adding it to the tap water. It was demonstrated that the leptospira were able to pass through an L3 Pasteur-Chamberland filter in sufficient numbers to be detected in the filtrate. With an L5 filter no organisms could be seen in the filtrate, though they appeared in ten days if it was incubated in a Petri dish.

Genus : Spirochæta Ehrenberg, 1834.

This genus was established by Ehrenberg (1834) for a long spiral organism which he found in water (Fig. 538), and to which he gave the name *Spirochæta plicatilis*. It is usually 200 to 500 microns in length and 0·5 to 0·75 micron in thickness. According to Zuelzer (1910), as explained

above, the body consists of an axial filament round which is wound in a spiral manner a protoplasmic thread (Fig. 539). The latter has rounded ends and is circular in a cross-section of the organism, which has the appearance of a ring of cytoplasm with the axial filament as a dot attached to the margin. There are no flagella, and the organism, revolving on its longitudinal axis, moves slowly over surfaces. Zuelzer states that the body is not limited by a definite membrane. Organisms of the dimensions given above have from 100 to 250 distinct turns, the distance between any two turns being about 2 microns. The axis of the spiral is not necessarily straight, but may show several irregular curves. The cytoplasm is of an alveolar nature, consisting of a tough portion in which are spaces filled with more liquid material. Deeply staining granules of either volutin or chromatin occur in that portion of the cytoplasm to which the axial filament is attached. Schaudinn (1905, 1907) stated that *S. plicatilis* possessed an undulating membrane and a nucleus in the form of a longitudinal filament. Subsequent investigations by Zuelzer and others have not confirmed these statements. The organism reproduces by transverse fission, a single long spirochæte dividing into two or into several smaller forms. Several other organisms which live in either fresh or salt water have been placed in this genus. Jaffé (1907) described an axial filament in a small spirochæte (*S. culicis*) which he discovered in larvæ of a species of *Culex*.

PARASITIC SPIROCHÆTES.

Under this heading will be considered a number of spirochætes which live in the bodies of animals, either as true parasites which give rise to definite disease, or as saprophytes which derive their nourishment from waste products. The majority of them have their counterpart in the free-living spirochætes, some of which, however, may be identical with pathogenic forms As explained above, the typical members of the genus *Spirochæta* are found in water, and it is doubtful if any of the parasitic forms have the same structure, though Schaudinn (1905) described and figured an axial fibre in *S. refringens* and Jaffé (1907) one in *S. culicis*. On this account the two genera *Treponema* and *Leptospira* are recognized, but it has to be remembered that the name can be applied to many free-living forms. The genus *Cristispira* stands apart, for none of the free-living spirochætes has yet been ascribed to this genus.

Genus: Cristispira Gross, 1910.

The best-known member of this genus is *Cristispira balbianii*, an organism which was discovered by Certes (1882) in the crystalline style which occurs in the stomach of oysters. Certes regarded the organism as

allied to the trypanosomes, and named it *Trypanosoma balbianii*. Gross (1910) first showed that it was not a trypanosome, and established the new genus *Cristispira* for its reception. It can be easily obtained for observation by removal of the stomach contents of fresh oysters. It is actively motile when first removed, and as it measures from 45 to nearly 100 microns in length and 1 to 1·5 microns in breadth it is readily observed (Fig. 540). Its most characteristic feature is the presence of a *crista*, which is attached to the body in a spiral manner. This is a fine membrane with a breadth of about that of the body itself. At the extremities of the organism the membrane narrows, and finally merges into the surface of the body. Gross did not observe a marginal filament on the free edge of the membrane, but Zuelzer believes that one is present. The organism is coarsely spiral, there being two to six broad turns. The cylindrical body, which has rounded ends, is limited by a definite membrane or periplast, while internally it has a chambered structure. There is a cylinder of tough cytoplasm surrounding a more liquid core, which is separated into segments by septa of the tougher cytoplasm, which pass across the organism at intervals. As Dobell (1911) points out, the tough cytoplasm has the arrangement of a bamboo with its nodes, the spaces in the bamboo corresponding with the more liquid cytoplasm (Fig. 537). According to Dobell, the only structures which can be distinguished are a series of small granules arranged in rings around the periphery of each septum. On account of their staining reactions, he believes that these granules are of chromatin nature. According to other observers, the granules are arranged in a more irregular manner throughout the tougher cytoplasm.

Fig. 539.—Diagrammatic Representation of Structure of *Spirochæta plicatilis*. (From Doflein, 1916, after Zuelzer.)

Reproduction takes place by transverse fission brought about by a constriction at one of the septa. When division is almost complete, the organism may become looped and the two portions which will form the daughter individuals may become twisted round one another, so that appearances suggesting longitudinal division are produced. Transverse fission is the method of reproduction which has been noted by most observers, and it can safely be

accepted that longitudinal division does not occur. Gross (1912), however, has stated that *Cristispira tapetos* of the mussel, *Tapes decussatus*, may break up into a number of small portions corresponding with the number of septa, and that each forms a kind of spore (Fig. 541). That this is a normal developmental process and that the spores can give rise to adult organisms again does not appear to have been demonstrated.

A number of other species which are parasitic in the crystalline styles of Mollusca have been placed in this genus. They differ from one another in size, in the character of the ends of the body, and other details.

Hollande (1921), as mentioned above, discovered in the small intestine of the guinea-pig a spiral organism 9 to 12 microns in length and possessing two or three large curves. The body is described as consisting of a fine
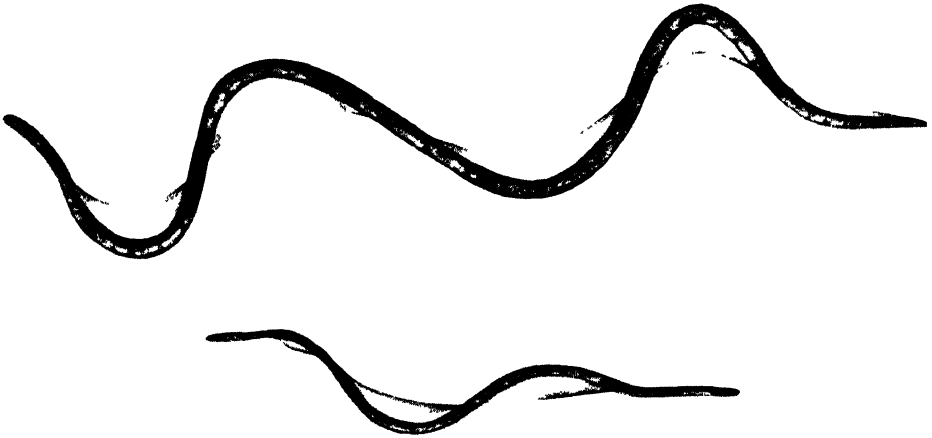
FIG. 540.—*Cristispira balbianii* FROM THE CRYSTALLINE STYLE OF THE OYSTER. FIXED IN SCHAUDINN'S FLUID AND STAINED WITH IRON HÆMATOXYLIN (× 2,000). (ORIGINAL.)

chromatic axial filament, but its most characteristic feature is a well-developed, flexible membrane which gives to the body a total breadth of 1·5 to 2 microns. Hollande considers it impossible to include this form in the genus *Cristispira*. He founds a new genus, naming the organism *Cristispirella caviæ*. He believes that a form discovered by Mesnil and Caullery (1916) in the intestine of a marine annelid worm, and named by them *Cristispira polydoræ*, probably belongs to the same genus.

Genus : Treponema Schaudinn, 1905.

The organisms which are included in this genus are so slender that accurate details of their minute structure are most difficult to obtain. According to some observers, certain forms possess membranes, or crista.

Thus Schaudinn stated that the organism known as *Spirochæta refringens* had a membrane, but other observers have failed to detect it. There appears to be no real evidence that a membrane is present in any one of the numerous species of *Treponema*. The flexible body is limited by a very fine but definite periplast, which encloses a homogeneous cytoplasm. The banded appearance which is sometimes seen in dried films stained by Romanowsky stain is probably artificial, and is no indication of a chambered structure like that occurring in *Cristispira balbianii*. Occasionally, with dark-ground illuminations, one or more refractile granules may be seen either on or in the spirochæte, and it has been suggested that these are spores. Some observers believe that a single organism may break up into a large number of minute spores, which may grow into the adult organisms. Definite terminal flagella are not present, but sometimes, under the dark field, a fine filament which may be spirally wound is observed at one or both ends of the organism. These are not constantly present, and they may be filaments of adherent material or the remains of the drawn-out periplast, which survives for some time after transverse division has taken place.

FIG. 541.—SUCCESSIVE STAGES IN SPORE FORMATION IN *Cristispira tapetos*. (FROM GONDER, 1920, AFTER GROSS. 1913.)

Though many observers have claimed that reproduction by longitudinal division occurs, it is now generally accepted that the only method of multiplication is by transverse fission. Appearances of longitudinal division arise when two organisms are closely wound round one another, or when the two portions of a transversely dividing one become similarly intertwined. The various species of *Treponema* agree with one another in possessing spiral bodies, the axis of which is a straight line in a condition of relaxation. Flexion and looping of the body may occur, but there is always the tendency in normal healthy individuals for the body to return to the position in which the axis is straight. In dried films various distortions occur, while the actual spirals are frequently obliterated. That this is a result of drying can readily be demonstrated by examining blood containing relapsing fever spirochætes with the dark field, and comparing the living spirochætes with those in dried and stained films of the same blood (Fig. 542). The living spirochæte has

the axis of the spiral body quite straight, unless it is temporarily bent owing to pressure against objects in the medium. In dried films spirochætes often appear as simple looped threads without the true spiral structure. If films which have been spread are exposed to osmic vapour for half a minute before being allowed to dry, the appearances after staining are much more like those of the living organisms. On this account, descriptions of spirochætes which are based on their appearance in dried films alone are most misleading, for the degree of distortion varies with the thickness of the film, the time it takes to dry, and other factors. As pointed out above, Zuelzer believes that the spirochætes which are included here in the genus *Treponema* have essentially the same structure as *S. plicatilis*, and that they should be placed in this genus. It seems safer at present to retain the genus *Treponema*, though future investigations may confirm Zuelzer's views.

The various species of *Treponema* differ from one another in the length and thickness of the body, the number of spirals occurring in any given length, and the character of the ends of the body, which may be rounded or tapering. Many forms which have been given specific names cannot be distinguished from one another morphologically. This is well illustrated by the relapsing fever spirochætes of man. Owing to the fact that relapsing fever in different parts of the world is transmitted by different arthropods and varies as regards its severity and number of relapses, it is claimed that a different species of spirochæte is responsible in each case. Animals which have recovered from one type of infection may not be protected against another type, and this fact is brought forward in support of the view that more than one species of *Treponema* is involved. African relapsing fever like that of Panama is transmitted by ticks, while other forms are conveyed by lice, and it is supposed that this difference justifies the separation of the African spirochæte as a distinct species. Whatever value may be attached to the variations in symptoms produced, the serological reactions, and the transmitting hosts, it remains a fact that there are no means of distinguishing the various named species of relapsing fever spirochætes from one another on morphological grounds, and it would be more logical to place them all in one species, and to regard the differences noted above as indications of separate races.

Spirochætes which are included in the genus *Treponema* are common inhabitants of all parts of the alimentary tract of man, and under certain conditions the bronchi also. They occur frequently around the orifice of the urethra. In certain chronic ulcerative conditions of the skin they are often numerous, and may extend deeply into the tissues. Many of these forms have been named, the distinctions being chiefly those of

size. It must be admitted, however, that there is little morphological ground for separating many of them. Thus the spirochætes which occur in the bronchi cannot be distinguished from some of those which live commonly in the mouth and pharynx, and these, again, are indistinguishable from many which are found in the intestine. The forms which occur in chronic ulcers of the skin have their counterpart in the spirochætes of the mouth. It is not an unreasonable hypothesis to suppose that there are one or more saprophytic spirochætes which can invade any part of the body, provided a suitable environment exists.

The spirochætes which produce syphilis and yaws have fairly definite characters, and usually can be recognized when the history of the case and the source of the material are known. But not infrequently there occur in the mouth saprophytic spirochætes, which cannot with certainty be distinguished from these definitely pathogenic forms. It is evident, therefore, that the various species of *Treponema* have been named, not on account of any definite morphological characters they possess, but because of their pathogenicity or non-pathogenicity, the types of infection they produce, and the situations in which they occur. At the present time it seems impossible to classify them in any other way, but it has to be recognized that species founded on these bases alone cannot be regarded as valid.

Spirochætes which Occur Chiefly in the Blood.

The species of *Treponema* which are included in this group are the various relapsing fever spirochætes of man and the similar forms which occur in the blood of animals. A certain number of these forms has been proved to be conveyed from host to host by arthropods, chiefly lice and ticks.

BLOOD SPIROCHÆTES OF MAN.—Relapsing fever is a disease which is characterized chiefly by repeated attacks of fever. An attack lasting from three to five days, during which the temperature remains elevated, is followed by an apyrexic period of similar length. Another attack occurs, again to be followed by a period of apyrexia. There may be as many as ten attacks alternating with periods of freedom from fever. Occasionally the disease is of a severe type, with marked jaundice and a condition approaching that of Weil's disease, to be described below (p. 1277).

The relapsing fever spirochæte was first seen by Obermeier (1873) in 1868, and was named *Protomycetum recurrentis* by Lebert in 1874. Cohn (1875) gave it the name *Spirochæta obermeieri*, by which it was known for many years. The name *Treponema*, proposed by Schaudinn (1905) for the spirochæte of syphilis, becomes the correct generic name for the

relapsing fever spirochæte, which is therefore *Treponema recurrentis* (Lebert, 1874).

Morphology.—The organism when observed alive is very active, and has the axis of its spiral nearly always straight (Fig. 542). It progresses with either end in front, and though it may perform bending movements, it tends to return to its normal condition. When an infection is abating as a result of the development of antibodies in the blood, variously coiled and clumped organisms may be found, but these are undoubtedly in a degenerating or dying condition. The movements and normal condition of a spirochæte can be seen with ordinary transmitted light, but more easily with dark-ground illumination. The average length of *T. recur-*
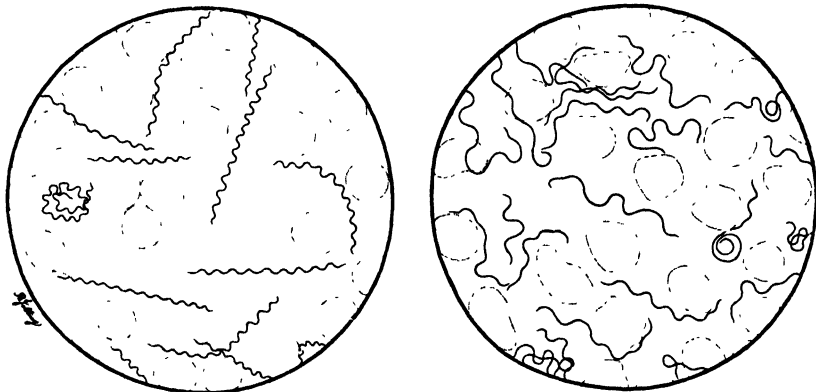
FIG. 542.—*Treponema recurrentis* (*T. duttoni*) OF AFRICAN TICK FEVER (× 1,000).
(ORIGINAL.)

A. Appearance of spirochætes as seen in the living condition.
B. Deformed spirochætes in a dry film stained by Leishman stain.

(Dotted rings represent red blood-corpuscles.)

rentis, as seen in the blood, is approximately 15 microns, the range in length of the majority of forms being 10 to 20 microns. Occasionally, shorter individuals occur, as also forms above 30 microns in length. The breadth is difficult to determine with accuracy, but is about 0·2 or 0·3 micron. The ends of the body are very slightly tapering. The number of spiral turns varies with the length of the organism. Each turn occupies from 2 to 3 microns, so that an organism 20 microns in length will show from seven to ten turns.

Reproduction is by transverse division. A constriction appears at the middle of the body, and the appearance of two spirochætes united by their tapering extremities is produced. As the daughter forms separate, a very fine thread, which probably represents the drawn-out periplast, may be seen to connect them. It may persist for some time, and owing to

the revolving movements of the spirochæte, it may itself become a spiral. The structure does not appear to be a flagellum, though after division is completed it may remain attached to the spirochæte, and resemble one.

The number of spirochætes in the blood of cases of relapsing fever varies from case to case. In some cases the field of the microscope will be crowded with them, while in others they are very scanty and difficult to find. They occur in the cerebro-spinal and other fluids of the body spaces, while they have been described as occurring in the urine. The latter statement undoubtedly requires confirmation, for it is not clear that the spirochætes which occur normally in and about the urethra were excluded. The spirochætes are present in the blood during the pyrexia, but they are absent, or present in very scanty numbers, between the attacks. It has been shown by several observers by inoculation of susceptible animals that the spirochætes are actually present in the blood of many cases during the apyrexic periods, and also for a considerable time after the last attack. Mackie (1907) succeeded in infecting a monkey by inoculating it with blood taken from a case in the apyrexic period when spirochætes were not demonstrable by microscopic examination. By similar inoculations of cerebro-spinal fluid it has been demonstrated by Plaut and Steiner (1920) that the spirochætes, which appear there as a rule shortly after the termination of the first attack, persist all through the disease and even after it has apparently terminated. They infected mice with cerebro-spinal fluid taken forty-five days after the last attack. The cessation of an attack and the disappearance of the organisms is associated with the appearance of antibodies in the blood. Towards the end of an attack, the spirochætes are often agglutinated in clumps and show various abnormalities.

The serum of individuals who have recovered renders them immune from further infection, has a definite action on the spirochætes, and will protect animals against inoculation. It was at one time thought that each type of relapsing fever spirochæte produced its own specific antibodies. Though this is to a large extent true, it is now known that different strains of one and the same type will produce different antibodies, so that it is impossible to regard the serological reactions as a reliable guide to the determination of species.

Species.—Owing to the fact that in different parts of the world the clinical picture of relapsing fever is not always the same, that in animals infections due to one type may not produce immunity against those due to another, and that the invertebrate vector may be a louse or a tick, there has been a tendency to regard the relapsing fever spirochætes as belonging to a number of different species to which the following names have been given:

T. recurrentis Lebert, 1874, Europe.
T. duttoni Novy and Knapp, 1906, West Africa.
T. novyi Schellack, 1907, America.
T. kochi Novy, 1907, East Africa.
T. carteri Manson, 1907, India.
T. rossi Nuttall, 1908, East Africa.
T. berberum Sergent and Foley, 1910, North Africa.
T. persicum Dschunkowsky, 1913, Persia.
T. ægypticum Mühlens, 1913, Egypt.
T. venezuelense Brumpt, 1921, Venezuela and Columbia.
T. neotropicalis Bates, St. John and, 1922, Panama.
T. hispanicum de Buen, 1926, Spain.

Some observers have believed that these various forms could be distinguished from one another morphologically. This is certainly not the case. The variations which may occur in any one of these supposed species are quite as marked as that which was presumed to indicate specific differences. Leishman (1907) stated that he could find no morphological difference between the European, Central African, and Indian strains, and that the supposed differences in virulence for laboratory animals did not exist. Attention was again called to this point by Macfie and Yorke (1917), who have shown that it is impossible to distinguish the same three strains by their morphological characters and dimensions. The various other distinguishing features which have been adduced, such as the difference in the type of the disease, especially the number of relapses, serological cross-immunity tests, and the variations in susceptibility of laboratory animals to inoculation, cannot be employed as reliable methods of differentiating species.

Serology.—Numerous investigations on the serum of men and animals who have recovered from infections have been made by Manteufel, Novy and Knapp, and many other later observers. Animals which have recovered from one strain may sometimes be infected with another, and it is largely on this account that the several species of relapsing fever spirochætes have been recognized. It seems, however, that the distinction is not of such specific value as was at one time maintained. The serum of recovered animals has an agglutinating and lytic effect on the spirochætes, and, moreover, is capable of preventing infection when injected at the same time as the infective blood. It is probable that the disappearance of spirochætes from the blood of relapsing fever cases is due to the appearance of antibodies, which, however, do not persist long enough to prevent relapse. Eventually, they become permanent and a cure associated with a lasting immunity results. Towards the end of an attack, auto-agglutination of the spirochætes in the peri-

pheral blood may occur. The agglutinating property of immune sera may still be manifest after a dilution of as much as one in two thousand, and Manteufel (1908) found that this property was specific and enabled him to distinguish the three forms — *T. recurrentis*, *T. duttoni*, and *T. novyi*. Novy (1907) came to the conclusion that the East African strain was serologically distinct from *T. duttoni* of West Africa, and named the organism *S. kochi*. Similarly, Sergent and Foley (1910) gave the name *Spirochæta berbera* to the North African form, though in its morphology and method of transmission it was identical with *T. recurrentis*. Brumpt (see Lavier, 1921) for similar reasons gave the name *S. venezuelense* to the relapsing fever spirochæte of Venezuela and Columbia. Bates and St. John (1922) have shown that the serum of patients and animals which have recovered from infections in Panama is specific as regards its spirochætocidal and protective action for this particular strain. They accordingly separate the Panama relapsing fever spirochæte as a separate species under the name *S. neotropicalis*. Cross-immunity tests with *T. venezuelense* and *T. neotropicalis* have not yet been applied. De Buen (1926) has given the name *T. hispanicum* to the tick-borne spirochæte of Spain.

Cunningham (1925), working with relapsing fever in Madras, states that the spirochætes which cause the first attack differ serologically from those which appear in the first relapse, but agree with those of the second relapse. There is thus an alternation of serological strains. The spirochætes during the first attack are inoculated to animals, and handed on directly from animal to animal. The same is done with those of the first relapse and second relapse. Serum from a man taken at the end of the first attack will cause agglutination of the spirochætes in the blood of animals containing the first attack strain or the second relapse strain, but not those in animals with the first relapse strain. On the other hand, the serum taken from the man at the end of the first relapse will agglutinate the spirochætes of the first relapse strain, but not the others. Similar observations had already been made by Levaditi and Roché (1907) and Jancsó (1918).

Several observers have reported positive Wassermann reactions in relapsing fever. Roaf (1922), for example, found that in eighteen cases a transient positive result was obtained in eleven. Its transient character distinguished it from the reaction of syphilis.

Transmission.—Though Cook (1904) had noted that relapsing fever occurred in Central Africa, and had described the spirochæte, it was P. H. Ross and Milne (1904) who first proved that the disease known as tick fever was a spirochætosis. This was confirmed by the independent observations of Dutton and Todd (1905) in the Belgian Congo. The latter

observers proved conclusively that the disease could be transmitted to monkeys by *Ornithodorus moubata*, and, furthermore, that the newly-hatched offspring of infective ticks were also capable of producing the disease (Fig. 543).

Möllers (1908) fed infected ticks on a series of monkeys at intervals of two months. A fatal infection was produced in the first eight monkeys and a non-fatal one in the next two. The two last monkeys of the series did not become infected. It is thus evident that ticks may remain infective for at least one and a half years. Möllers also noted that when spirochætes had once been ingested by ticks, infection was able to pass through the eggs to the third generation.

In Somaliland and Abyssinia the tick *O. savignyi*, which also occurs in Arabia and India, is almost certainly the vector of relapsing fever. It has been suspected of transmitting the disease in Angola by Wellman (1905). Brumpt (1908*a*) succeeded in infecting a monkey in Paris with a batch of these ticks received from Somaliland. The causative spirochæte is referred to as *Sp. abyssin* by Mesnil (1908). The Central and West African strain (*T. duttoni*) was also transmitted from monkey to monkey by Brumpt through the agency of *O. savignyi*. Working in Panama, Bates, Dunn and St. John (1921) proved by experiments on man, monkeys, and rats that *O. talajæ* is responsible for the spread of the relapsing fever spirochæte (*T. neotropicalis*). In Venezuela and Columbia the transmitting host is *O. venezuelensis*, as first demonstrated by Tejera (see Brumpt, 1922). Brumpt

FIG. 543.—*Ornithodorus moubata* (♀) THE TRANSMITTER OF *Treponema recurrentis* IN CENTRAL AFRICA (× 5). (ORIGINAL.)

was also able to infect animals in Paris with ticks sent to him from Venezuela. As a result of immunity tests, Brumpt (see Lavier, 1921) concluded that the spirochæte was a distinct species, and gave it the name *T. venezuelense*.

A disease which has been long known as mianeh occurs in certain parts of Persia. Schneider (1908) called attention to the fact that *Argas persicus* and *O. tholozani* were found in certain districts, and that the inhabitants attributed a serious disease to their bites. It was considered probable that this was relapsing fever, which is now known to be common

in Persia. Dschunkowsky (1912) observed the spirochæte, and proposed to call it *Spirochæta persica*. The vector is probably *O. tholozani*, suspected by Dschunkowsky, rather than *A. persicus*, with which Sergent and Foley (1910) and other observers have failed entirely to transmit the human spirochæte (Fig. 549). So far no accurate experiments have been made with *O. tholozani* nor with *O. lahorensis*, which has also been regarded by Harold (1922) as a probable transmitter of relapsing fever in Persia. The method by which *O. moubata* brings about infection is doubtful. When the tick feeds, fluid exudes not only from the anus, but also from the coxal glands which occur in the first segments of the limbs. Spirochætes have been demonstrated in the coxal fluid, and it is supposed that this contaminates the wound inflicted by the tick. It is possible that the salivary gland secretion as also the fluid exuded from the anus is infective.

Though the tick had been definitely incriminated as a vector in Central Africa and Central and Southern America, it was evident that other arthropods were responsible in those parts of Europe, India, North America, and North Africa, where suitable ticks do not occur. Nevertheless, Manteufel (1908) and Neumann (1909) were able to transmit the Russian strain (*T. recurrentis*) and the American strain (*T. novyi*) from rat to rat by means of *O. moubata* imported to Europe, though both strains are normally transmitted by lice. Recently, however, de Buen (1926) has shown that *A. marocanus*, or a nearly allied species, is the vector in Spain.

Mackie (1907a), observing an epidemic of relapsing fever in a settlement of boys and girls in India, concluded that the spirochæte (*T. carteri*) was conveyed by lice (Fig. 544). There were more cases amongst the boys than the girls, who were less infested with lice than the boys. Spirochætes were present in the intestine, body cavity fluid, and organs of the lice, and also in fluid pressed from the mouth parts. Transmission experiments were not successful. Similar observations were made by Sergent and Foley (1908) in Algeria. They succeeded in infecting a monkey with spirochætes (*T. berberum*) by inoculating a crushed-up louse which had been removed from a human case six days before. The same observers gave a more detailed account of their observations in 1910, but obtained no conclusive evidence to incriminate the louse except that two women who had slept in the lousy bedding of a case of relapsing fever and on whom lice from cases were placed contracted the disease. Smith (1909) came to the conclusion that relapsing fever in Egypt was a louse-borne disease.

The exact mechanism of infection by lice was elucidated by Nicolle, Blaizot and Conseil (1912), working in Tunis. They failed entirely to convey the disease by the bites of lice, and noted that the spirochætes taken into the stomach disappeared entirely in five to six hours. After

the lapse of about eight days, spirochætes appeared in the body-cavity fluids. At this period the material obtained by crushing lice in saline solution produced infection when injected into monkeys and when placed on the conjunctiva or excoriated skin of human beings. They concluded that infection was spread by scratching, during which material from damaged infected lice was brought into contact with scratches on the skin or conveyed on the fingers to the conjunctiva. It was also demonstrated that exceptionally the offspring hatched from eggs laid by infected lice were also infective. The number of actual transmission experiments made with lice does not appear to be numerous, though it is quite evident that the relapsing fever of Europe, Asia, North Africa, and North America, where lice alone are associated with the disease, is transmitted by these
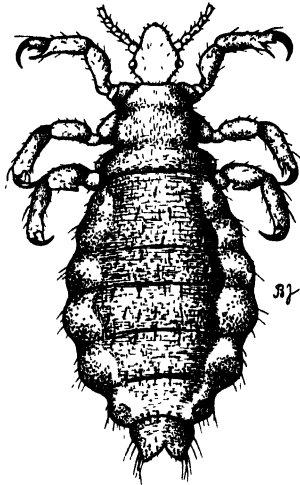
FIG. 544.—*Pediculus vestimenti* (♀) THE TRANSMITTER OF *Treponema recurrentis* (× 13·5). (AFTER PATTON AND CRAGG, 1913, SLIGHTLY MODIFIED.)

arthropods. Selwyn-Clarke, le Fanu, and Ingram (1923) have transmitted the relapsing fever spirochæte of the Gold Coast by means of lice, which are undoubtedly the vectors in this locality. Manteufel (1908) showed that the European virus (*T. recurrentis*) could be transmitted from rat to rat by the agency of the rat louse (*Hæmatopinus spinulosus*), while Neumann (1909) proved this for the African virus (*T. duttoni*).

Many observers have attempted to transmit infection by means of bed bugs. These experiments have invariably failed, though it has been shown that the spirochætes may persist in the stomach of the bug for as long as thirty days. The writer (see G. U. Smith, 1909, p. 36) observed a persistence of living spirochætes in the bug for twenty days. Nuttall (1908) proved that the spirochæte in bugs remained virulent for animals for at

least five days, while Dunn (1923) has shown that rats can be infected by inoculating them with macerated bugs thirty-two days after ingesting spirochætes. Mackie (1907) succeeded on one occasion in infecting a monkey by exposing it to the bites of bed bugs which had fed on an infected monkey immediately before; and Nuttall (1908) infected a mouse by transferring to it thirty-five bugs which had just partially fed on an infected mouse. Such direct transmission is probably comparable with the method of infection demonstrated by Mackie (1907), who infected monkeys by pricking them with a syringe needle which had been inserted into the skin of infected monkeys. Dunn (1923) failed to effect transmission by bugs fed on infected men or animals one to thirty-six days previously. Experiments conducted with arthropods other than ticks and lice have invariably failed to transmit human relapsing fever.

Development in the Invertebrate.—The most straightforward account of the behaviour of relapsing fever spirochætes in the invertebrate host is that given by Nicolle, Blaizot and Conseil (1912 and 1913), and by Nicolle and Lebailly (1920). These observers studied the North African spirochæte (*T. berberum*) by making smears from lice at different intervals after feeding on infected blood, by examination of the tissues and fluids by dark-ground illumination, and finally by fixing entire lice, staining them by Levaditi's silver nitrate method and cutting serial sections (Fig. 545). It was found that the spirochætes disappear entirely from the stomach during the course of the first day after feeding. They at first become immobile, and finally disintegrate. At the anterior part of the stomach and in the œsophagus, the spirochætes apply themselves to the surface of the lining cells, and many of them penetrate the cytoplasm of the cells. This stage is, however, of short duration, as no trace of spirochætes can be found in the stomach or in the cells after twenty-four hours. The behaviour of the intracellular forms could not be traced, owing to the number of granules which occur naturally in these cells. From the time of the disappearance of the spirochætes at the end of the first twenty-four hours to the sixth day, no trace of spirochætes could be found anywhere in the lice. From the sixth day onwards, however, spirochætes begin to appear in the body-cavity fluids, and, gradually increasing in number, they spread to all parts of the body, including the legs and antennæ. They are still present in the body-cavity fluids on the nineteenth day. Sergent and Foley (1914), who confirmed these observations, observed spirochætes in the body-cavity fluids as late as the twenty-fifth day. According to Nicolle and his co-workers, the forms which appear are distinctly narrower and finer than those originally ingested. The organisms, after their disappearance from the lumen of the stomach and the lining cells, never reappear in this situation, and no information was obtained as to what happens to

them during the interval between their disappearance from the stomach and reappearance in the body cavity. The method of conveyance of infection to man is by damage caused to lice on the skin. These insects are easily hurt, the exceedingly delicate legs being readily broken off. When this occurs, the body-cavity fluid escapes on to the skin, and infection of the wounds inflicted by the louse, or by scratching, takes place. It is pointed out that a human being who had allowed himself to be bitten from 30,000 to 40,000 times by infected lice never became infected, as care had been taken not to damage the insects. As already remarked,

1   2   3

FIG. 545.—SECTIONS OF LICE STAINED BY SILVER NITRATE METHOD AFTER FEEDING ON BLOOD INFECTED WITH THE RELAPSING FEVER SPIROCHÆTE (× ca. 450). (AFTER NICOLLE AND LEBAILLY, 1920.)

1. Stomach of louse fixed six hours after first feed on infected blood and immediately after a second feed on uninfected blood. Epithelial cells containing granules of blood-pigment and spirochætes lying on surface of cells and between them.
2. Leg of louse nine days after feed on infected blood. Spirochætes in the intramuscular spaces.
3. Antenna of same louse as in 2.

it was found that only occasionally did the infection pass through the eggs of lice to the succeeding generation.

As regards the course of development of spirochætes in ticks, there are two views. Dutton and Todd (1907), working with *T. duttoni* and *Ornithodorus moubata*, first called attention to the breaking up of the spirochætes into granules, which occurred in numbers in the Malpighian tubes, and they suggested that the granules possibly represented a method of reproduction. Leishman (1918, 1920) likewise maintains that the spirochætes ingested by the ticks break up into granules, which are able to

reproduce by simple or multiple division. These granules, which are spherical, rod-shaped, or comma-shaped, occur particularly in the Malpighian tubes and ovaries. They often occur in clumps, which sometimes appear to be enclosed by a membrane. They are supposed to represent the phase of development of the spirochætes at low temperatures. When the ticks are kept at a temperature of 34° to 37° C., the granules are said to elongate, and finally become transformed into spirochætes, which render the ticks infective. By the use of the dark field Leishman (1920) believes that he has obtained evidence of the growth of spirochætes from the granules (Fig. 546). Similar granules were described as stages of development of the chicken spirochæte, *T. anserinum* (*T. gallinarum*), in *Argas persicus* by Balfour (1911), Fantham (1911), and Hindle (1912). According to the second view, the granules do not represent a phase in the development, but are derived from disintegrated spirochætes, or belong to the tissues of the tick. Wittrock (1913) stated that he was able to find the granules in uninfected as well as infected specimens of *O. moubata*. They also occurred in the larvæ hatched from eggs laid by uninfected adults. Wittrock also proved that ticks were already infective within an hour of feeding on blood containing spirochætes, and that they remained continuously infective for ninety days. Spirochætes were at first difficult to find in the body-cavity fluid, but became more numerous after the lapse of several weeks. He came to the conclusion that there is a continuous but slow multiplication of the spirochætes at low temperatures. At higher temperatures reproduction is more rapid. Kleine and Eckard (1913), as also Gonder (1914), expressed similar views. The former found that ticks were not infective if spirochætes could not be demonstrated in the fluids of the body. Spirochætes occurred in the eggs of the ticks, and it was concluded that the eggs were invaded by actual spirochætes, and not by any other form. Ticks which contained only the granules or comma forms of Leishman were unable to produce infection. Koch (1905) had already noted that spirochætes taken up by *O. moubata* did not multiply in the intestine, from which they quickly disappeared. Later, they appeared in the body cavity, and could be found in large numbers about the ovaries. Spirochætes were also demonstrated by Koch within the eggs which had been laid. In newly-laid eggs they were scanty, but they increased rapidly by active multiplication, till tangled masses of spirochætes were present. The observations of Koch were confirmed by Carter (1907).

It is difficult to form a definite opinion as to the true nature of the granules described by Leishman, who claims to have seen stages which suggest the growth of the granules into spirochætes. In dealing with such minute objects which assume various shapes and forms, it is exceedingly

difficult to be sure that a series which is traceable in smears actually represents an evolution of granules into spirochætes. It has also to be remembered that similar granules have been found by Wittrock in ticks which, as far as can be judged, were free from spirochætal infection. On the other hand, there is the definite fact that the spirochætes disappear from the stomach and intestine, and only later become recognizable in the body-cavity fluid and tissues of the ticks. It is just as reasonable to assume that they are present, but are at first too few in number to be detected, as to suppose they occur in granule form. It does not seem
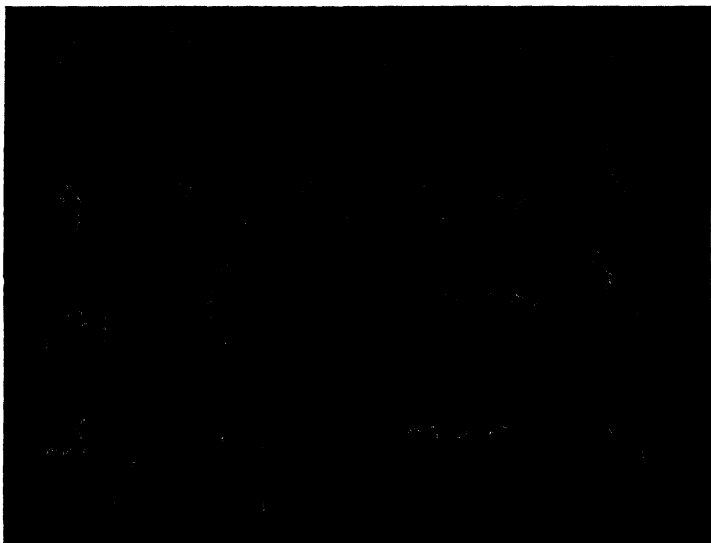
FIG. 546.—DARK-GROUND APPEARANCE OF TISSUES OF *Ornithodorus moubata*, SHOWING APPARENT TRANSFORMATION OF GRANULES INTO SPIROCHÆTES (× 2,000). (AFTER LEISHMAN, 1918. FROM *Tropical Diseases Bulletin*, vol. xii., p. 209.)

1-6. Granule clumps enclosed by membrane.
7-10. Commencing growth of spirochætes from granule clumps.
11-17. More advanced stages in growth.
18-21. Double growth from granule clumps.

impossible that the granules of Leishman and other observers are of the nature of *Rickettsia*, which have been found to occur in various arthropods, and which have been dealt with above. It is an undoubted fact that many of the spirochætes ingested by ticks degenerate, and in so doing 'may break up into granules, but that granules actually grow into spirochætes again has not been proved. That spirochætes have a granule or spore phase in their life-history is not impossible, but lacks demonstration. It seems to the writer more reasonable to assume for the present that the spirochæte in the body of both its vertebrate and invertebrate host

remains as a spirochæte, and multiplies continuously by transverse fission, and that when it is apparently absent in material proved to be infective to animals, this is due, not to its having assumed some other form, such as that of a granule, but to its numbers being so scanty that it escapes recognition. It is safer to accept this view till absolute proof of the existence of a granule phase has been obtained.

Susceptibility of Animals.—Of experimental animals, the monkey is most susceptible to inoculation from human beings. The course of the infection is similar to that in man, and there may occur one or more relapses. Rats and mice are less readily infected from man, though frequently a transient infection lasting about twenty-four hours is produced. These animals are more readily infected after a strain has been passed into the monkey. Spirochætes appear in the blood in about two or three days, and persist for two or three days longer, when they disappear. Though relapses in these animals are of rare occurrence, the blood may still be infective for many days after this. Todd (1920) showed that the heart blood of rats could still produce infection when injected into other rats as long as thirty-five days after the apparent disappearance of spirochætes. The writer inoculated a mouse with *Trypanosoma cruzi* thirty days after it had recovered from a spirochætal (*T. duttoni*) infection. With the appearance of trypanosomes in the animal, the spirochætal infection of the blood recurred.

When once an infection has been established in mice or rats, it may be kept indefinitely in these animals by sub-inoculations. It has been claimed that animals are more readily infected with *T. duttoni* than other supposed species, but there is no real ground for this assertion. Individual strains of one and the same species vary considerably in their virulence for animals.

Fraenkel (1907) proved that mice could be infected by allowing them to feed on the tissues of infected mice. Manteufel (1908) infected animals by placing infected blood on the intact skin, while Nattan-Larrier (1909) showed that infection readily took place by introduction of blood into the mouth, rectum, vagina, and conjunctiva, as well as on to the shaved skin of rats.

Culture.—Though it has been noted that relapsing fever spirochætes will survive for many days in blood diluted with various fluids, only a few observers have been able to maintain a strain by continued sub-culture. Noguchi (1912) cultivated various strains in the following manner: A piece of rabbit kidney is placed in a test-tube and a few drops of infected blood are then added, after which about 15 c.c. of ascitic or hydrocele fluid are introduced. Growth took place at 37° C. Sub-cultures were obtained in a similar manner. Hata (1913) simplified this method. He used horse serum diluted with twice its volume of physiological saline solution. Test-tubes containing a requisite quantity of the mixture are heated in the

water bath at a temperature rising slowly from 58° to 71° C., at which they are kept for thirty minutes. When cool, a piece of the buffy coat on the surface of the horse blood-clot or a piece of kidney is pushed through the semi-solid medium to the bottom of each tube. In this medium at 37° C. the maximum growth of spirochætes is obtained on the fifth to the seventh day. More recently, Kligler and Robertson (1922) have been successful with a medium made as follows: Horse or rabbit serum is diluted with one or two parts of saline solution or undiluted ascitic fluid. To each 10 c.c. of this fluid is added 1 c.c. of a 10 per cent. peptone broth. The reaction is adjusted to pH=7·2, and 3 to 4 c.c. of the mixture are placed in each test-tube, which is inoculated with a drop of blood or 0·1 c.c. of fluid from a previous culture, and the surface is covered with a layer of oil. In the case of the sub-cultures, a drop of rabbit's blood may with advantage be added just before the layer of oil. The tubes are incubated at 28° to 30° C. Aristowsky and Höltzer (1924) report that they have passed a strain through 200 sub-cultures during the course of a year and a half. The medium used was prepared by adding 8 c.c. of saline solution to 4 c.c. of the serum of a young horse in a test-tube, and then introducing a piece of blood-clot or white of a hard-boiled egg. The tubes were inoculated, and then incubated at 35° C. It was necessary to sub-culture every forty-eight to seventy-two hours.

Growth in these media is slow, the spirochætes never becoming so numerous as they do in cultures of leptospira.

Passage through Filters.—Novy and Knapp (1906), as well as other observers, have shown that the relapsing fever spirochæte may pass through the pores of a Berkefeld filter. Blood diluted with ten parts of saline-citrate solution was filtered under a pressure of 50 pounds. The filtrate was infective to rats. Breinl (1907) observed that *T. duttoni* in the tissues of experimental animals broke up into granules, and he and others have supposed that it is these forms which pass through the filter and render the filtrate infective. Todd (1920) repeated this experiment, as other observers have done. In two cases spirochætes were actually seen in the filtrate, so that there is no reason to suppose that its infectivity was due to any hypothetical granular stage of the organism.

Action of Drugs.—Infections with *T. recurrentis* are readily controlled by intravenous injections of salvarsan or allied arsenic compounds. A single suitable dose will cut short the disease in such a way that relapse does not occur in the majority of cases. No other drugs have this marked effect upon relapsing fever spirochætes.

BLOOD SPIROCHÆTES OF CATTLE.—Theiler in the Transvaal was the first observer to discover spirochætes in the blood of cattle. The

organism was studied by Laveran (1903) in films sent him by Theiler, and he named it *Spirillum theileri*. It measures 20 to 30 microns in length, and closely resembles the relapsing fever spirochætes of man. The same organism was seen by Ziemann (quoted by Lühe, 1906) in a calf in the Cameroons, Koch (1905) in German East Africa, Lingard (1907) in India, Schein (1910) in Annam, Carpano (1912) in Eritrea, and Blieck (1913) in Java. Heanley (1906) saw a very similar spirochæte in the blood of buffaloes in China. The organism seen by Nuttall (1910) in blood-films made from a buffalo (*Bos caffer*) of East Africa, and which was named by him *Spirochæta bovis caffris*, is undoubtedly an intestinal form which had contaminated the blood-films.

Theiler (1905) and Dodd (1906) showed that *T. theileri* was directly inoculable from ox to ox, and also from ox to sheep. The natural transmission is by means of ticks, as demonstrated by Theiler (1905 and 1909). *Margaropus decoloratus*, a one-host tick, ingests spirochætes which pass into the egg, so that the newly-hatched larvæ are infected. *Rhipicephalus evertsi*, which does not remain on one host, acquires infection through the egg, like *M. decoloratus*, but is also infective in the adult stage through the nymphs having fed on infected animals (Fig. 429). With ticks (*M. decoloratus*) received from Theiler, Laveran and Vallée (1905) were able to infect cattle in France. Koch (1905) stated that he had followed the development of the spirochæte in ticks as far as invasion of the egg. Brumpt (1919), working in France, was able to infect cattle with *T. theileri* by feeding ticks (*M. australis*) which had been sent to him from Brazil. According to Theiler, the spirochætes are usually present in the blood of infected animals in small numbers only, and give rise to no symptoms. When the cattle are ill with other infections, such as piroplasmosis, large spirochætal infections may occur.

BLOOD SPIROCHÆTES OF HORSES.—The spirochæte which occurs in the blood of horses was discovered by Theiler (1904) in the Transvaal. It was named *Spirochæta equi* by Novy and Knapp (1906). Dodd (1906) showed that the spirochæte was inoculable to sheep and cattle, and concluded that it was identical with *T. theileri*. The same organism was seen by Martin (1906) and by Stordy (1906) in British East Africa. Carpano (1912) observed it in Eritrea, Petzoldt (1915) in German East Africa, and Velu (1916) in Morocco. Velu stated that he had been able to infect rats, dogs, rabbits, and even fowls. The spirochæte of the horse resembles *T. theileri* of cattle, and, as suggested by Dodd, is probably the same species.

Trautmann (Mühlens, 1913) saw a spirochæte in the blood of a donkey in German East Africa, an observation also made by Mason (1916) in Egypt.

BLOOD SPIROCHÆTES OF SHEEP.—The spirochæte of the sheep was first seen by Theiler (1904) in the Transvaal. Dodd (1906), as a result of inoculation experiments, came to the conclusion that it was identical with *T. theileri* of cattle. The same organism was seen by Martoglio and Carpano (1904) in Eritrea, and was named *Spirochæta ovina* by Blanchard (1906). It was also found by Lühe (1906) in films made in the Cameroons and by O'Brien (Simpson, 1914) in the Gold Coast. Macfie (1916), who saw spirochætes in blood-films of sheep and goats in the Gold Coast, states that, as his films were made at the slaughter-house, it is not impossible that the films had been contaminated from the intestine. From the character of the spirochætes, this seems a very probable explanation of their presence in the blood-films.

BLOOD SPIROCHÆTES OF PIGS.—No one appears to have found spirochætes of the relapsing fever type in the blood of pigs. King, Baeslack and Hoffmann (1913), under the name of *Spirochæta suis*, described an organism from the blood of pigs suffering from swine fever. It measured 5 to 7 microns in length and 1 micron in breadth. Arnheim (1914) saw what was possibly the same spirochæte. Numerous authors have recorded spirochætes from the intestine of pigs and in intestinal ulcers, and the highly improbable suggestion that these are the cause of swine fever has been made. It is possible that the blood forms are really the intestinal spirochætes. Macfie (1916) found spirochætes in the blood of slaughtered pigs on the Gold Coast, but it seems very probable that these and similar forms seen in films made from slaughtered cattle, sheep, and goats were contaminations from the intestinal contents. It is remarkable with what ease blood becomes contaminated with intestinal organisms if the intestine has been damaged before films are made from the blood of an animal which has been opened.

BLOOD SPIROCHÆTES OF OTHER MAMMALS.—Lingard (1907) recorded spirochætes of the relapsing fever type from the blood of camels and elephants in India. Bruce, Hamerton, Bateman. Mackie and Lady Bruce (1911) observed a spirochæte in the blood of a bush buck (*Tragelaphus scriptus*) in Uganda. It measured 9 to 15 microns in length, and was inoculable to white mice. It was named *Spirochæta brucei* by Brumpt (1913). A very similar form was seen by Todd and Wolbach (1912) in the blood of a roan antelope (*Hippotragus equinus*) in the Gambia. The forms seen by Ross, P. H. (1907), in blood films made from antelope which had been shot were probably intestinal contaminations.

Monkeys have been extensively employed in experimental work in human relapsing fever, to which they are more susceptible than other animals. Naturally occurring spirochætal blood infections of monkeys have been observed on several occasions. Castellani and Chalmers (1910)

described as *Spirochœta macaci* a spirochæte seen by them in *Macacus* sp. in Colombo. It resembled the relapsing fever spirochæte of man in India. Thiroux and Dufougeré (1910) observed a similar form in *Cercopithecus patas* of Senegal, and named it *S. pitheci*, while Ranken (1912) saw one in *C. ruber* in the Sudan. It was inoculable to mice, in which animals the strain was maintained by sub-inoculations. Plimmer (1912) saw a similar form in a monkey (*C. sabœus*) which had died in the Zoological Gardens in London.

Spirochætes have been recorded from the blood of many smaller mammals. Mathis and Leger (1911) observed a form of the relapsing fever type in rabbits in Tonkin. It measured 14 to 17 microns in length, and was named *S. raillieti*. A similar form was described by Gasperi (1912) from the guinea-pig.

A small organism 2 to 9 microns in length and possessing two to six spirals was obtained in culture in defibrinated blood containing glucose by Macfie (1914) from a guinea-pig in West Africa.

The small North African rodent, *Ctenodactylus gundi*, was found by Nicolle (1907) occasionally to harbour a spirochæte of the same type, which was named *S. gondii*. Leger, A. (1917, 1918), observed a spirochæte of the relapsing fever type in the small rodent, *Crocidura stampflii*, of Dakar in West Africa, and gave it the name *S. crocidurœ*. Later, Leger, M. (1924) observed what appeared to be the same organism in *Rattus norvegicus*, *Rattus coucha*, and *Golunda campanœ* of the same locality, and suggested the possibility of these rodents acting as reservoirs of the spirochæte of human relapsing fever. As *Ornithodorus moubata* does not occur in this locality, some other vector must be responsible for the transmission. The form seen in *C. stampflii* was studied by Leger, A., and Le Gallen (1917), who found that it was inoculable to rats, mice, and monkeys, in which it behaved like the human relapsing fever spirochæte.

Nicolle and Comte (1905, 1906) discovered a spirochæte 12 to 18 microns in length in the bat, *Vespertilio kuhli*, in Tunis. The infection was transmissible by blood inoculation to uninfected bats. Novy and Knapp (1906) proposed the name *S. vespertilionis* for this organism. It was again seen by Gonder (1907) in the same bat, and by Coles (1914) in *Vesperugo pipistrellus* in England.

Bosselut (1925) describes a spirochæte of the relapsing fever type in the blood of a dog in Algiers. He names the organism *Spirochœta canina*.

In connection with rat-bite fever, attention will be called to the occurrence of small organisms which are probably spirilla in the blood of rats and mice. These forms have been seen by a number of observers (p. 1285). The one described by Prowazek (1907) as *S. lutrœ* from the blood of an otter is very similar, as also that recorded by Carpano (1912) from the blood of a marmot of Eritrea.

BLOOD SPIROCHÆTES OF BIRDS.—Sakharoff (1891) observed spiro-chætes in the blood of sick geese in the Caucasus, and regarded them as pathogenic. He named the organism *Spirochæta anserina* (Fig. 547). Marchoux and Salimbeni (1903) described a similar disease of fowls in South America, and demonstrated its transmission by *Argas persicus.* The spirochæte of fowls was referred to as *Spirochæta gallinarum* by Stephens and Christophers (1904).

Spirochætosis of fowls has now been found to occur in many parts of the world. The disease in geese has been less commonly seen. It
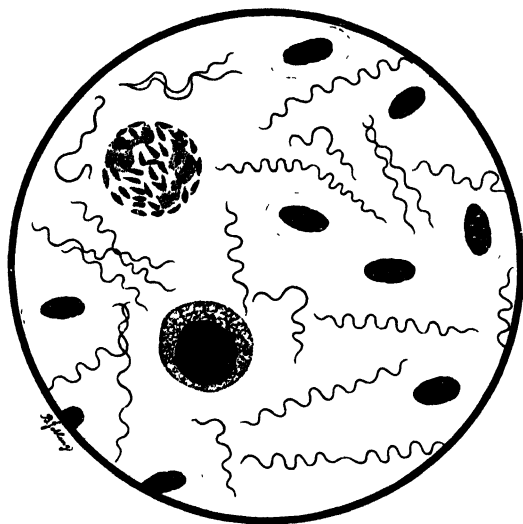
FIG. 547.—*Treponema anserinum* FROM THE BLOOD OF A FOWL (× 1,000). (ORIGINAL.)
An eosinophile leucocyte, a mononuclear leucocyte, and several red blood-corpuscles are shown.

occurs also in ducks, as noted by Parrot (1920), who suggests the name *Spirochæta anatis* for the organism. Mason (1916) records spirochætal infections of fowls, ducks, and geese in Egypt.

As with the relapsing fever spirochætes of man, many investigations have been conducted with strains of the spirochæte found in fowls, with the result that numerous specific names have been given. Thus, Brumpt (1909) proposed the name *S. neveuxi* for a strain from Senegal and (1909a) *S. nicollei* for one from Tunis. Balfour (1911) suggested the name *S. granulosa penetrans* for the Sudan form, because of certain granular structures occurring in the red blood-corpuscles of infected fowls, which he regarded as possibly representing developmental stages of the spirochætes. Neumann and Mayer (1914) gave the name *S. gallinarum* var. *hereditaria* to a North African strain studied by Hindle (1912) on

account of the fact that the infection passed through the eggs of the tick to the third generation. There seems to be very little doubt that the various spirochætes described from fowls, geese, and ducks belong to one species, and no reliance can be placed upon the immunity test as a means of separating species. The correct name for the spirochæte is *Treponema anserinum* (Sakharoff, 1891). Morphologically, it closely resembles the relapsing fever spirochæte of man. It is very doubtful if the two forms can be distinguished morphologically.

Fowls which have been inoculated with infected blood or exposed to infected ticks first show spirochætes in the blood in two to four days. Death may occur four or five days later, when the blood contains enormous numbers of spirochætes, which are often agglomerated in masses towards the end of the disease. In some cases the disease runs a more chronic course, and fowls may survive two or more weeks. As it progresses, the fowls become increasingly weak, emaciated, and anæmic, till finally they are unable to stand, and death follows.

Susceptibility of Birds and other Animals.—The organism is readily inoculated from fowl to fowl, and also to geese, ducks, turkeys, canaries and other small birds. Pigeons are not so easily infected, though Gerlach (1925) successfully infected one bird. Levaditi (1904) succeeded in producing a temporary infection in rabbits by injecting large quantities of blood into the peritoneal cavity, and in conjunction with Lange (1905), by using very young rabbits, was able to hand on the infection from one animal to another. Deutz (1912) succeeded in infecting mice, but the spirochætes remained in the blood for not more than seventy-two hours. By direct inoculation from bird to bird the virulence of a strain increases considerably, as pointed out by Montgomery (1908).

Like the relapsing fever spirochæte of man, *T. anserinum* can be cultivated, as first demonstrated by Noguchi (1912a).

Possible Intracellular Stage.—As regards the development of *T. anserinum*, reproduction by binary fission is the only method which is certainly known. The advocates of the " infective granule theory " believe that under certain conditions the spirochæte may break into granules which can again develop into spirochætes, as explained above for the relapsing fever spirochætes of man. Balfour (1907) drew attention to curious bodies which occur in the red blood-corpuscles of Sudanese fowls (Fig. 548). He later discovered that these bodies were associated with the presence of spirochætes in the blood, and came to the conclusion that they were probably derived in the first place from spirochætes which had penetrated the red cells. No satisfactory evidence of the actual penetration of a cell by a spirochæte and its transformation into a granule was obtained. It was concluded that the granule, which has the appearance of a

coccus, increased in size and became a group of granules, the process being supposed to represent a multiplication by schizogony. It seems to the writer that the possibility of these granular bodies being extruded portions of the nuclei resulting from the profound changes which occur in the blood during the course of an infection has not been excluded. In apparently healthy birds the separation of small portions of the nuclei of the red blood-corpuscles appears to be of regular occurrence, and it is possible that this may be exaggerated in the case of fowls suffering from spiro-chætosis. Hindle (1912) came to the conclusion that this was the explana-tion of the granules described by Balfour. In view of the fact that the
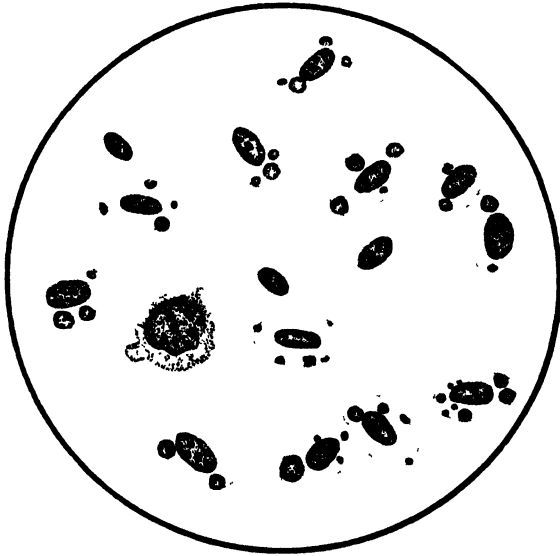
FIG. 548.—INTRACORPUSCULAR BODIES OCCURRING IN ASSOCIATION WITH SPIRO-CHÆTOSIS OF FOWLS IN THE SUDAN (× 1,000). (AFTER BALFOUR, 1909.)

intracorpuscular bodies did not occur in fowls infected with the spiro-chæte in other localities, such as South America, Balfour (1914) suggested that in the Sudan, and also South Africa, where Jowett (1910) had noted a similar condition, there were possibly two distinct infections—one caused by the spirochæte, and the other by an intracorpuscular parasite—which ran concurrently in the fowls. The fact that Franchini (1924a) in Italy has seen what appear to be identical bodies in the blood-corpuscles of a bird (*Hypoleis hypoleis*) without any associated spirochæte infection rather sug-gests that this explanation may be the correct one. If the bodies actually represent a distinct parasite, it is difficult to associate it with any of the known intracorpuscular parasites, none of which it appears to resemble. Dschunkowsky and Urodschevitch(1923) have noted the same bodies in

Macedonian fowls suffering from spirochætosis. Gerlach (1925) failed to find them in Austria, where fowl spirochætosis occurs.

Transmission.—Marchoux and Salimbeni (1903) were the first to demonstrate the transmission of the fowl spirochæte by *Argas miniatus* in South America. Balfour (1907) suspected the tick *A. persicus* as being the transmitting agent of fowl spirochætosis in the Sudan, while Reaney (1907) made a similar claim for the disease in India (Fig. 549). Brumpt and Foley (1908) succeeded in infecting fowls with *A. persicus* collected twenty-three to thirty-five days before in an endemic focus of the disease in North Africa. Galli-Valerio (1908) shortly afterwards published an account of the successful infection of fowls in Switzerland by means of ticks sent from Tunis. This tick is now known to be the vector throughout the Old World. In Australia, Dodd (1910) and Gilruth (1910) discovered the disease in fowls, and demonstrated its transmission by ticks, which were supposed to be *A. persicus*. Other ticks are also capable of transmitting the spirochæte experimentally, but that they do so in nature is doubtful. Schellack (1908) incriminated *A. reflexus*, an observation confirmed by Williamson (1908) in Cyprus. Fülleborn and Mayer (1908), Brumpt (1908), and Neumann (see Neumann and Mayer, 1914), demonstrated that *Ornithodorus moubata* could also convey infection. Mayer (1914) transferred a number of mites from canaries infected with *T. anserinum* to healthy canaries. In a few days the birds became infected, so that it is evidently possible for mites to play the part of transmitting host. Gerlach (1925) has succeeded in transmitting the disease from fowl to fowl by means of the mite *Dermanyssus avium* in Austria. He believes that the mite is the chief vector in this locality.

FIG. 549. —*Argas persicus* (♀), THE CHIEF TRANSMITTER OF *Treponema anserinum* (× 8). (ORIGINAL.)

Development in the Tick. — The development of *T. anserinum* in *A. persicus* has been extensively studied. The infection will pass through the egg to the next generation, and, as demonstrated by Hindle (1912), without reinfection this generation may hand it on to the next. There is the same difference of opinion as to the behaviour of *T. anserinum* in *A. persicus* as there is for the relapsing fever spirochætes of man in *O. moubata*. Balfour, in his studies, which extended from 1907 to 1913,

observed the breaking up of the spirochætes into granules and the presence of granules in large numbers in the tissues of the ticks, which, though infective, appeared to be free from spirochætes. Hindle (1912) gave a description and diagram of what he considered to be the life-cycle of *T. anserinum* in *A. persicus* and in the fowl (Fig. 550). The tick is supposed to inoculate granules into the body of the fowl or to contaminate the puncture wound with granules in its excreta. These grow into spirochætes, which reproduce by fission in the usual manner. A spirochæte may also break up into granules, which may again become spirochætes. The spirochætes taken into the tick invade the cells of the body, especially those of the Malpighian tubes, and in these, as also in the lumen of the gut, they break into granules, which multiply till very large numbers are produced. It is these granules which Hindle supposes are inoculated into the fowl or invade the egg of the tick to produce infection of the succeeding generation. Hindle found that if ticks which had fed on infected blood were kept at a uniform temperature of 28° C. the spirochætes disappeared in a few days, being replaced by the coccoid bodies or granules, which occurred chiefly in the cells of the Malpighian tubes and ovaries, as also in the lumen of the gut. If a batch of ticks in this condition were incubated at 37° C., it was found that spirochætes appeared in the cœlomic fluid and later in all the organs of the body. By the examination of ticks at different intervals Hindle came to the conclusion that he had traced the elongation of the coccoid bodies and their final transformation into spirochætes. Similar changes were detected in eggs incubated at 37° C. These observations were made with stained films only, and it is possible that though the coccoid bodies actually elongated at the higher temperature, their transformation into spirochætes was only apparent, and resulted from a rapid multiplication of a few spirochætes which were present. It seems that the absolute proof that the coccoid bodies were stages of the spirochæte, or that they developed into spirochætes was not obtained. Marchoux and Couvy (1913) studied the infection in *A. persicus*, and came to the conclusion that the granules in the ticks were structures which occurred in this tick and other arthropods quite apart from the presence of spirochætes. Furthermore, they stated that when a tick had once taken up spirochætes these are constantly present, though sometimes so fine in starving ticks that special staining methods have to be employed for their demonstration. On this account it is never possible to be sure of inoculating fowls with material containing granules alone. According to Marchoux and Couvy, the spirochætes enter the salivary glands, and when they cannot be found in the tissues of the ticks they are still present in the salivary ducts. They also state that when eggs are laid they are coated with fluid from special secretory glands.

This fluid contains spirochætes, which penetrate the eggs, even passing through their chitinous envelopes. A single egg may contain as many
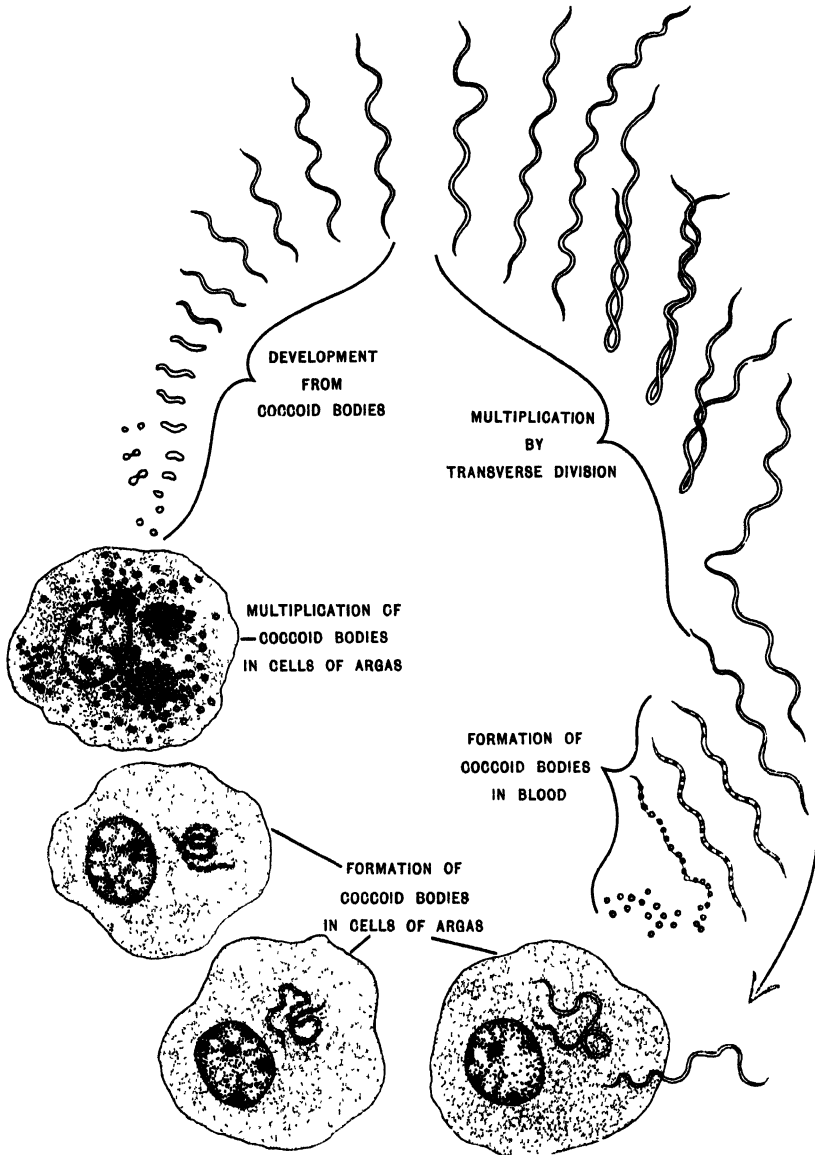
FIG. 550.—LIFE-CYCLE OF *Treponema anserinum* IN THE BLOOD OF THE FOWL AND IN THE TICK (*Argas persicus*), ACCORDING TO HINDLE. (AFTER HINDLE, 1911; from *Parasitology*, vol. iv., p. 475.)

as thirty spirochætes. Hindle has informed the writer that he also has found spirochætes in the salivary duct of ticks which have been kept at a temperature of about 27° C.

It seems safe to conclude, therefore, that even though some of the granules in the ticks may have been derived from the spirochætes, there is no conclusive evidence that they can again develop into spirochætes. It does not seem improbable that the spirochæte remains in the tick in the spirochæte form, that it multiplies by fission as it does in the blood of the fowl, and that it is actually in this form that it infects the fowl, and invades the eggs of the tick to lead to infection of the next generation.

Töpfer, quoted by Mühlens and Hartmann (1906), cultivated a spirochæte from the blood of the little owl (*Athene noctua*), while Franchini (1924) has described a spirochæte infection of snipe (*Gallinago gallinago*) in Italy. The organisms, which were numerous in the blood, were 10 to 18 microns in length.

BLOOD SPIROCHÆTES OF COLD-BLOODED VERTEBRATES.—Spirochætes have been described from the blood of reptiles by several observers. Dobell (1910) saw a spirochæte of the relapsing fever type in the Ceylon snake, *Tropidonotus stolatus*. He named it *Spirochæta tropidonoti*, while Neiva, da Cunha and Travassos (1914) described as *Treponema tropiduri* a form seen by them in the South American snake, *Tropidurus torquatus*.

Spirochætes have been found in fish, especially marine forms, by several observers. Dutton, Todd and Tobey (1906) described as *S. jonesii* a spirochæte-like organism from the African fish, *Clarias angolensis*. As there is difficulty in obtaining uncontaminated blood from fish, it is possible this was not actually a blood spirochæte. Neumann (1909*a*) described two forms (*S. gadi* and *S. pelamidis*) from the marine fish, *Gadus minutus* and *Pelamys sardi*. Henry (1910 and 1912), and Duboscq and Lebailly (1912 and 1913) described spirochætes to which they gave various specific names from a number of marine fish of which spirochætal infections appear to be of common occurrence.

Spirochætes which Occur Chiefly in the Tissues.

Certain spirochætes which live in tissues, such as *Treponema pallidum*, are definitely parasitic, while others which occur in the superficial parts of chronic ulcers are merely saprophytic. It is not improbable that the latter are intestinal forms which have contaminated the lesions, while the possibility of their origin from free-living organisms has to be considered. The whole question of the relationship of these saprophytic spirochætes to free-living and intestinal forms requires investigation.

TISSUE SPIROCHÆTES OF MAN.—Syphilis, Yaws, and Rabbit Syphilis.— Of the spirochætes which can be regarded as tissue-invading forms the most important is *Treponema pallidum*, the cause of syphilis, which

is handed on from one person to another by contact of infected surfaces. It has essentially the same structure as the relapsing fever spirochætes, from which it differs only in being smaller and in having a larger number of coils for any given length. There exists a whole series of spirochætes showing every grade of transition between *T. pallidum* and *T. recurrentis*. Some of the spirochætes which occur in the mouth are structurally identical with *T. pallidum*, as also certain free-living spirochætes found in water and described by Zuelzer (1921).
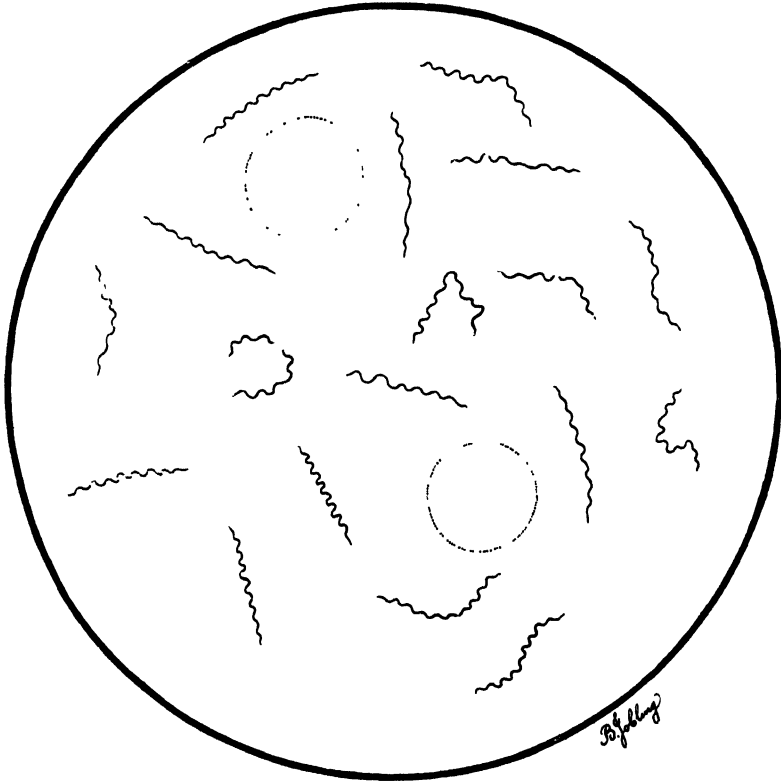
FIG. 551.—*Treponema pallidum* IN SMEAR FROM A SECONDARY SYPHILITIC LESION (× 2,000). (ORIGINAL.)

T. pallidum, as it occurs in human tissues, varies in length from 5 to 15 microns (Fig. 551). It is exceedingly fine, being barely ·25 microns in breadth. A form of intermediate size has about ten spiral turns, which towards the centre of the organism are slightly broader and closer together than at the extremities, which may be represented by portions which show no spiral turns. On account of its fineness and small size, prolonged staining of very thin films with good Romanowsky stains is necessary to colour it satisfactorily. It can readily be demonstrated in

films and tissues by silver nitrate methods, but is best seen with dark-ground illumination (Fig. 552). The organism has been demonstrated in all stages of syphilis, though in tertiary lesions and in the tissues of cases of general paralysis it is present in small numbers only.

An organism which is morphologically indistinguishable from *T. pallidum* was described by Castellani (1905, 1905a) in the lesions of yaws (Fig. 553). He (1905) suggested for this organism two names—*Spirochæta pertenuis* and *S. pallidula*—the first of which, being the first mentioned, will give the correct specific name. Both *T. pallidium* and *T. pertenue* are very susceptible to salvarsan and allied drugs. Salts of bismuth are only a little less active, while those of mercury are able to eradicate infections, especially in the tertiary stage.

Arzt and Kerl (1914) drew attention to a venereal disease of rabbits, in the genital lesions of which they discovered an organism which appeared to be morphologically identical with that of syphilis. Organisms of a similar type had been noted previously in rabbit lesions by Ross (1912) and Bayon (1913). The disease, which had been studied by Schereschewsky (1920), Klarenbeek (1921), Levaditi, Marie and Isaïcu (1921), Warthin, Buffington and Wanstrom (1923), and others, is readily communicable to healthy rabbits, but other animals, including monkeys, are not susceptible to inoculations. Rabbits which have recovered from the infection are still susceptible to inoculations with *T. pallidum*. Noguchi (1921a) suggested the name *T. cuniculi* for the organism occurring in the spontaneous disease of rabbits.

FIG. 552.—SECTION OF LIVER OF CHILD WITH CONGENITAL SYPHILIS, SHOWING *Treponema pallidum* (SILVER NITRATE STAIN) (× 800). (AFTER SOBERNHEIM FROM KOLLE AND WASSERMANN'S HANDBUCH, 1913.)

With the organism of syphilis there have been carried out numerous serological investigations which have culminated in the well-known Wassermann test for the disease. This test is dependent upon what is termed " fixation of the complement," owing to the fact that complement,

which is present in normal blood, combines with antigen—an emulsion of the organism, an extract made from it, or some substitute—in the presence of the antibody produced by the organism. The test is made by adding to a mixture of antigen and complement the serum which may or may not contain antibody (amboceptor). If antibody be present—*i.e.*, if there is an infection—the complement will be fixed. Fixation of the complement is detected by adding to the mixture of the three substances red blood-corpuscles and homologous hæmolytic serum. If the complement be free, the red blood-corpuscles are visibly hæmolysed.

FIG. 553.—*Treponema pertenue* IN A SECTION OF A LESION IN EXPERIMENTAL YAWS IN THE CHIMPANZEE (SILVER NITRATE STAINING) (× 1,000.) (AFTER NATTAN-LARRIER, 1913.)

Ulcus tropicum. — This somewhat ill-defined disease is characterized by a chronic ulceration of the skin, especially of the lower limbs, which affects persons in tropical countries. Though spirochætes, frequently associated with fusiform bacilli, had been found by numerous observers in chronic ulcerations of the skin, it was Prowazek (1907), and later Keysselitz and Mayer (1909), who attempted to define the disease as an ulceration caused by a spirochæte named by Prowazek *Spirochæta schaudinni*. The range of dimensions of this organism is given as 6 to 35 microns, the majority of forms measuring 12 to 25 microns, so that in size they correspond with the relapsing fever spirochætes (Fig. 554). As regards the spirals, most of the descriptions have been based on the appearances in dried films, which, as pointed out above, obliterate the natural turns present in the living organisms. The figures show forms which are merely fine filaments, and others with spirals like those of the relapsing fever organisms. It is probable that in the living condition *Treponema schaudinni* is structurally very similar to *T. recurrentis*. In sections of a number of ulcers which the writer examined it could be seen that the surface of the ulcer contained spirochætes, fusiform bacilli, and other organisms. A little deeper there was a layer of spirochætes and fusiform bacilli, while, deeper

still, spirochætes alone were found. There seems to be no real evidence that the spirochæte is actually the primary cause of the condition.

Vincent's Angina.—In this inflammatory condition of the tonsils and pharynx spirochætes and fusiform bacilli have long been known. Blanchard (1906) gave the name *Spirochæta vincenti* to the spirochæte. A very similar association of spirochætes and fusiform bacilli occurs in hospital gangrene, and the conditions in which these organisms are found are
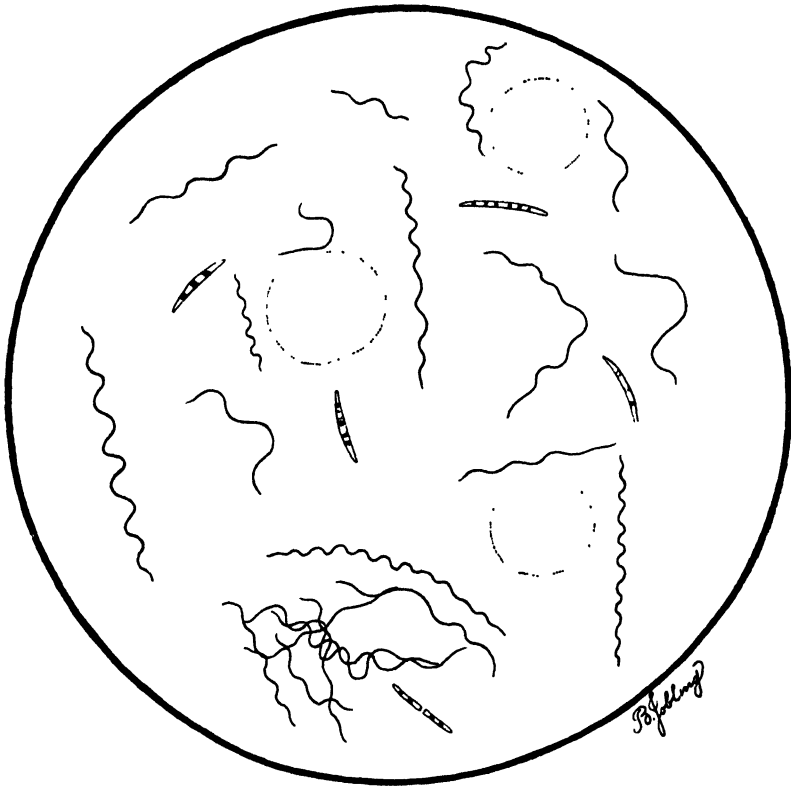
Fig. 554.—Spirochætes and Fusiform Bacilli in a Smear from a Tropical Ulcer (×2,000). (Original.)

Dotted rings represent red blood-corpuscles.

now commonly known as Vincent's disease. It is very probable that the organisms described from tropical ulcers are of the same nature, and that *Treponema schaudinni* of Prowazek is identical with *T. vincenti* (Fig. 554). Furthermore, it is not improbable that the spirochæte is merely a saprophytic organism, and may be identical with some of the species of *Treponema* which live in the mouth and alimentary canal.

The organism described by Cleland (1909) as *Spirochæta aboriginalis,*

from *Granuloma venereum*, is merely a saprophytic form of the same type. Several observers have described spirochætes as occurring in carcinoma, while White and Pröscher (1907) claim to have seen spirochætes (*Spirochæta lymphatica*) in the glands in cases of Hodgkin's disease, lymphatic leukæmia, and lymphosarcoma.

TISSUE SPIROCHÆTES OF ANIMALS.—Numerous observers have described spirochætes in the tissues of animals. King, Baeslack and Hoffman (1913) noted a short spirochæte which they named *Spirochæta suis* in intestinal ulcers of pigs. Similar forms have been found in intestinal and other lesions in many animals. There does not seem to be any reason to regard these forms as pathogenic. It seems not improbable that ulcers or other lesions in man and animals are liable to invasion with saprophytic spirochætes, which may be merely those occurring naturally in the intestinal tract. This is probably the explanation of *Treponema refringens* and other forms which occur in association with *T. pallidum* in syphilitic lesions.

Spirochætes of the Alimentary, Respiratory, and Genito-Urinary Tracts.

FORMS IN MAN AND OTHER VERTEBRATES.—The whole of the alimentary tract of man and animals is liable to harbour spirochætes. Sometimes these saprophytes are present in enormous numbers, and various pathogenic properties have been ascribed to them. It is impossible to consider here the numerous species which have been described. Some of them are minute organisms very similar to *Treponema pallidum*, while others are larger, and resemble *T. recurrentis*. It is probable that there are several species living in this situation, but as many of them have been described from ordinary dried films, few details of their true morphology are available. The forms which the writer has seen with dark-ground illumination have the typical spirochæte structure, and though there is considerable variation in size, it is very difficult to form an opinion as to where one species begins and another ends. In dried films the true picture of the living organism is nearly always lost. Of the mouth organisms (Fig. 555) there appear to be three main types: small forms (*T. dentium* Koch, 1877) very like *T. pallidum*, large forms (*T. buccalis* Cohn, 1875) like *T. recurrentis*, and intermediate forms (*T. medium* Hoffman and Prowazek, 1905). The first of these is probably identical with Noguchi's *T. macrodentium*, and the second with his *T. microdentium*, both of which he obtained in pure culture from the mouth in cases of pyorrhœa. Similar forms occur in the intestine (Fig. 556), and though these have been given distinctive names, there is reason to believe that they may be identical with the mouth

spirochætes. Werner (1909) gave the name *Spirochæta eurygyrata* to an intestinal form which had an **S**-shaped body, and measured 6 microns in length. Another form of similar dimensions, and possessing two to six spirals, he called *S. stenogyrata*. A longer form, more of the relapsing fever type, was named *S. intestinalis* by Macfie and Carter (1917). An organism of similar type found in a male urethra was named *S. urethræ* by Macfie (1917), while another smaller form from the vagina was called *S. vaginalis* by the same observer (1916).
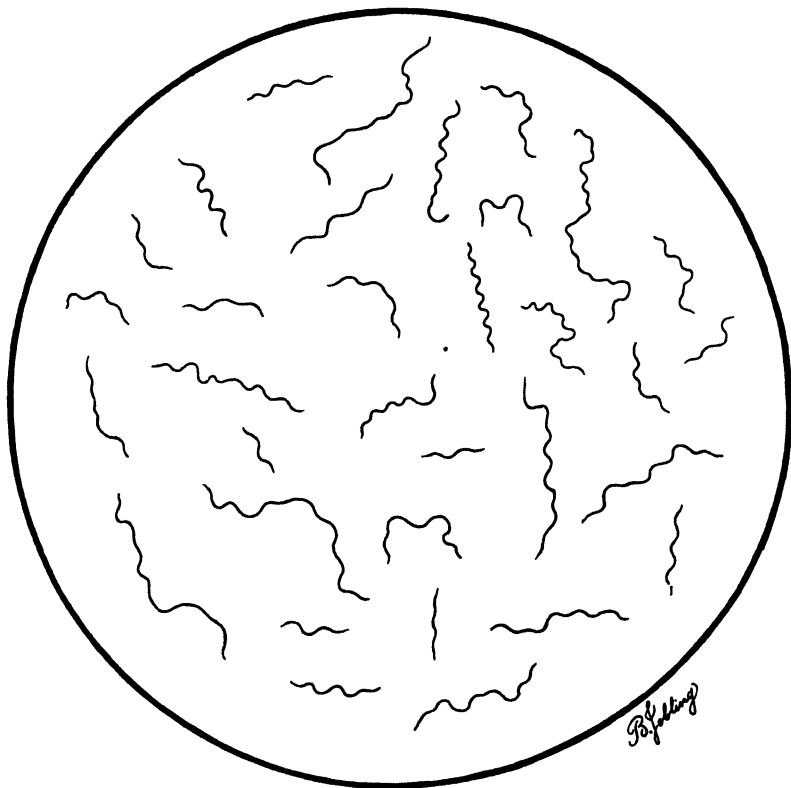
FIG. 555.—SPIROCHÆTES IN A SMEAR OF MATERIAL FROM A HUMAN MOUTH (× 2,000). (ORIGINAL.)

The forms which occur in the mouth may spread to the bronchi, and can be found in sputum coughed up from the lungs. Castellani (1906, 1909) described a disease, bronchial spirochætosis, which he supposed was due to a particular spirochæte. Castellani and Chalmers (1910) give the name of the organism as *S. bronchialis* Castellani, 1907, but the writer can find no mention of this name in the reference quoted. It appears that the name was first published by Castellani in 1909. Since Castellani first described the condition numerous observers have reported it from many

parts of the world, and have noted as clinical types almost every conceivable form of pulmonary disorder. These records merely demonstrate that spirochætes are more commonly present in the bronchi than was supposed, but they do not supply any evidence that the spirochætes are actually pathogenic. It would be equally logical to describe as buccal spirochætosis every abnormal condition of the mouth when spirochætes can be discovered there. There is very little evidence that
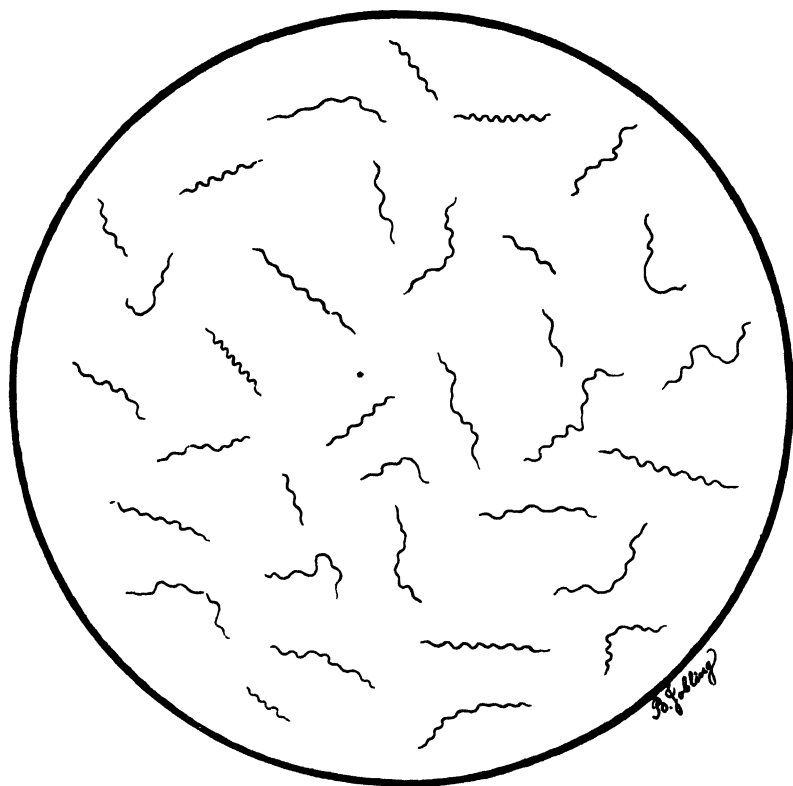
FIG. 556.—SPIROCHÆTES IN A SMEAR OF MATERIAL FROM A HUMAN INTESTINE (× 2,000). (ORIGINAL.)

the spirochætes which occur in the bronchi are in any way more pathogenic than those which are found so commonly in the mouth. Vincent (1922) believes that in certain cases the bronchial spirochætes are the same as those which occur in Vincent's angina, but it has not been conclusively demonstrated that spirochætes are the actual cause of this condition. The spirochætes which occur in sputum coughed up from the lungs in certain inflammatory conditions resemble very closely the various species of *Treponema* which occur in the mouth, and in spite of the numerous claims which have been made, it is impossible to distinguish

Spirochæta bronchialis from other previously named oral forms. Etchegoin (1923), examining the blood-stained sputum of tuberculous patients, found spirochætes in fourteen out of fifteen cases examined. Similar forms have been described from the nose, and others commonly occur about the orifice of the urethra, and may contaminate the urine.

The difficulty of classifying these saprophytic spirochætes is well illustrated by the observations of Hogue (1923), who has cultivated pure strains of *S. eurygyrata* in a medium made by adding 0·3 c.c. of dilute pig serum (serum 1, water 3) to 15 c.c. of 0·85 per cent. saline solution. Small drops of fluid were placed on a cover-glass till one was obtained in which only a single spirochæte occurred. The drop was then introduced into the medium, which was incubated at 35° C. The resulting spirochætes, grown from a single individual, showed forms varying in length from 4 to 56 microns, with a corresponding variation in the number of turns which themselves varied considerably in arrangement. It is evident that many of the spirochætes described as distinct species are merely stages of development of one form. Parr (1923), who has investigated the intestinal spirochætes of man, comes to the conclusion that until precise data regarding the cultural characters of pure strains are available it is better to regard all the intestinal forms as belonging to the single species, *S. eurygyrata*. Delamare and Achitouv (1924), who have cultivated intestinal spirochætes in a serum bouillon, have come to a similar conclusion.

Recently Kermorgant (1925) claims to have proved that mumps is caused by a spirochæte which can be cultivated from the saliva in the early stages of the disease. The spirochæte grows anaerobically in rabbit serum in symbiosis with a motile bacterium. The cultures are said to reproduce the disease in its typical form, including complications such as orchitis, when inoculated to monkeys. Kermorgant does not appear to have demonstrated the organism in the parotid gland, where it would be expected to occur, though he has isolated it from the glands of experimentally inoculated animals by cultivation. He supposes that a granule phase of the spirochæte has a part in its life-cycle.

The alimentary tracts of animals similarly harbour spirochætes of various forms, and it is safe to conclude that they are universally distributed.

FORMS IN INVERTEBRATES.—Spirochætes are commonly present in the intestine of insects. Novy and Knapp (1906) observed a spirochæte in smears of the intestinal contents of *Glossina palpalis*. They named it *Spirochæta glossinæ*. Sergent, Ed. and Et. (1906), noted spirochætes in the intestine of larvæ of *Anopheles maculipennis*. Jaffé (1907) described as *S. culicis* a form seen by him in the larva of *Culex* sp.,

while Léger and Duboscq (1909) record one from the larva of *Ptychoptera contaminata*. Schaudinn (1904), who saw spirochætes in the intestine of *Culex pipiens*, believed them to be developmental stages of *Leucocytozoon ziemanni* of the little owl. Porter (1910) saw spirochætes (*S. melophagi*) in *Melophagus ovinus*, while Dobell (1912) gave the names *Treponema stylopygæ* and *T. parvum* to two forms seen by him in the intestine of cockroaches. Laveran and Franchini (1920) also saw spirochætes in these animals. Patton (1912) recorded *S. ctenocephali* from the dog flea (*Ctenocephalus felis*), and Chatton (1912) *S. drosophilæ* from *Drosophila confusa*. Noc and Stévenel (1913) found spirochætes in the intestine of *Aëdes* (*Stegomyia*) *argenteus*, while Noc (1920) saw them in the Malpighian tubes of the same mosquito. Pringault (1921) found a new species, *S. phlebotomi*, in the intestine of *Phlebotomus perniciosus*. Zuelzer (1925) has given the name *Spirochæta noelleri* to a form occurring in the intestine of larvæ of *Simulium noelleri*. Prowazek (1910) found spirochætes in the intestine of white ants (*Termes lucifugus*) in Japan. He named the organism *Spirochæta minei*. A form seen in white ants in Ceylon was named *S. termitis* by Dobell (1910), while Doflein (1911) gave the name *S. grassii* to a form he had seen in termites in Italy. Spirochætes have also been described from fresh-water molluscs. Gonder (1908) gave the name *S. hartmanni* to a form in species of *Pinna*, and Schellack (1909) the name *S. pusilla* to one occurring in species of *Unis*, *Lima*, *Ostrea*, and *Tapes*.

Laveran and Franchini (1921) described a spirochæte from the intestine of lygeid bugs living on euphorbias. A similar form was found in the latex of the plants, and it is concluded that there is a spirochætosis of plants which is transmitted by the bugs.

Genus : Leptospira Noguchi, 1917.

The spirochætes of this genus differ little from the forms which have been considered as belonging to the genus *Treponema*. The body, however, possesses a large number of spirals, which are more closely wound than the spirals of a typical *Treponema*. When seen alive with the dark-ground illumination (Fig. 557, B), the first impression is that of a long, flexible filament which travels across the field while bending and straightening out in various ways. The ends of the organism are frequently bent into the form of a crook, which during the rapid revolutions gives rise to an appearance of a bulbous extremity. Careful observation, however, reveals the numerous fine spiral turns. Zuelzer (1921) believes that its structure is essentially that of *Spirochæta plicatilis*, and that there is a similarly arranged axial filament, but other observers have not been able to detect the filament. It must be admitted that the typical members

of the genus are easily recognized when seen by dark-ground illumination. In the examination of fluids containing blood it has to be remembered that various filaments or " pseudospirochætes " may occur as a result of changes in the red blood-corpuscles, as first described by Addison (1861). These frequently bear a striking resemblance to leptospira, but they are not so actively motile as living leptospira, do not have the closely-wound spiral structure, and cannot be satisfactorily stained in dried films (Fig. 557, A).

Weil's Disease.—This is a frequently fatal disease characterized by a sudden onset of high fever, albuminuria, intense jaundice, and hæmorrhages. In severe cases the resemblance to yellow fever is very

A   B
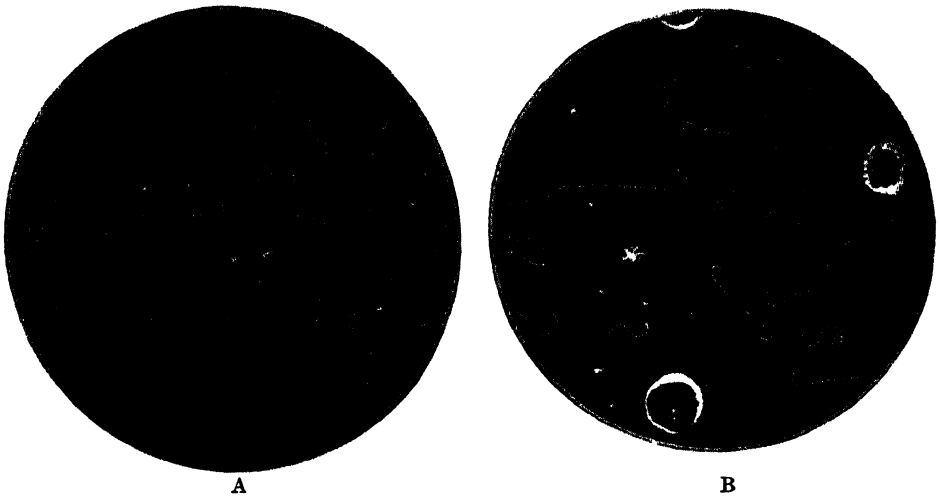
FIG. 557. –LEPTOSPIRA-LIKE ARTEFACTS AND TRUE LEPTOSPIRA AS SEEN BY DARK-GROUND ILLUMINATION (× 1,000). (A, AFTER KNOWLES AND DAS GUPTA, 1924; B, AFTER WENYON: from *Tropical Diseases Bulletin*, vol. xxi., p. 282.)

A. Granules and blood-filaments in blood. The filaments appear to arise from the red blood-corpuscles.   B, *Leptospira icterohæmorrhagiæ* from culture.

close. One attack, lasting about a week, may be followed by a second one three or four days later. The causative organism was first seen in Japan by Inada and Ido (1915), who named it *Spirochæta icterohæmorrhagiæ*. Huebener and Reiter (1915) discovered the organism in Germany, and proposed for it the name *S. nodosa*, while Uhlenhuth and Fromme (1916) gave it the name *S. icterogenes*. There is no doubt that the various strains isolated from human beings and rats in different parts of the world belong to the one species first described by the Japanese. Noguchi (1917) placed the organism in the new genus *Leptospira*. According to him (1925) it varies in length from 8 to 24 microns. A form 10 microns in length has about twenty close spiral turns (Figs. 558 and 559).

The organism occurs in the tissues, especially the liver and kidneys, and also in the blood and urine of cases of Weil's disease (Fig. 560). It may be discovered in the blood or tissue juices by direct observation with the dark ground, in films stained with Romanowsky stain, or tissues prepared by Levaditi's method. Guinea-pigs are very susceptible to inoculation, and as a rule rapidly die of a fatal infection. They react by showing a high temperature, marked jaundice, and hæmorrhagic condition of the lungs, glands, and abdominal organs. When the spirochætes cannot be found by direct observation, diagnosis may be made by inoculating guinea-pigs, which are very susceptible to infection, with blood, urine, or material obtained by puncture of the liver. Rabbits are less easily infected, while monkeys, cats, chickens and pigeons, and larger animals are still more resistant.

It is a remarkable fact that wild rats are commonly found to have a naturally acquired infection with *L. icterohæmorrhagiæ*. In a series of London rats examined by Stevenson (1922), 30 per cent. were found to be

FIG. 558.—*Leptospira icterohæmorrhagiæ* STAINED BY SILVER NITRATE. (×*ca.* 5,000). (AFTER MARTIN AND PETTIT, 1919.)

harbouring the organism. There appeared to be no seasonal incidence, as some observers have described. The spirochæte can be found in the urine, kidneys, and liver of rats by dark-ground illumination or in stained smears of tissues. It can also be isolated by culture or by inoculation of material, such as the fluid from crushed kidney tissue, into guinea-pigs, in which it gives rise to the condition seen after inoculation with the human virus.

It appears that the disease may occur in dogs, for Krumbein and Frieling (1916) noted that certain dogs which had associated with two human beings suffering from Weil's disease were in a similar condition.

The presence of leptospira was not demonstrated in these animals, but Courmont and Durand (1917) proved that dogs were susceptible to inoculation with *L. icterohæmorrhagiæ*. Uhlenhuth and Fromme (1919) examined a dog suffering from a disease resembling Weil's disease, and demonstrated leptospira in the organs. Křiváček (1924) claims to have found leptospira in the organs of seventeen of twenty-one dogs suffering from " canine typhus " in Germany. There exists in England a " malignant jaundice" of dogs, and Okell, Dalling and Pugh (1925) have isolated from dogs suffering from this disease a leptospira which is highly pathogenic to guinea-pigs, and appears to be identical with the organism of Weil's disease. Cultures of the organism reproduce the original disease when inoculated into dogs. Dunkin and Laidlaw (1926) have discovered *L. icterohæmorrhagiæ* in a sick and jaundiced fox in England.

There is no definite evidence of the natural method of transmission of *L. icterohæmorrhagiæ*, though its presence
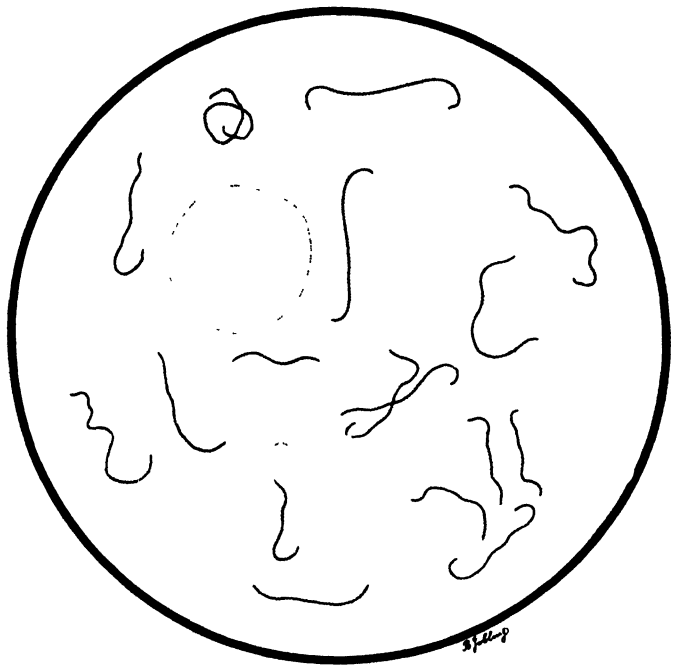
FIG. 559. — *Leptospira icterohæmorrhagiæ*, AS SEEN IN A GIEMSA-STAINED SMEAR OF THE KIDNEY OF A NATURALLY INFECTED RAT (× 2,000). (ORIGINAL.)
The dotted ring represents a red blood-corpuscle.

in the urine has suggested the possibility of human beings acquiring the disease from food or water contaminated by the urine of rats, while Zuelzer's observation that a strain of *Leptospira* cultivated from tap water proved pathogenic to guinea-pigs affords further evidence of a water origin of the disease. Buchanan (1924) has also infected guinea-pigs with leptospira from the slime on the roof of a mine in Scotland where cases of Weil's disease occurred. Symptoms and lesions resembling those produced by the inoculation of the human and rat strains resulted.

The organism is easily cultivated in serum of rabbits or other animals which has been diluted with ten times its volume of physiological saline solution or in semi-solid agar to each test-tube of which ten to twenty drops of rabbit's blood have been added.　After repeated sub-culture the organism tends to lose its virulence for guinea-pigs (see pp. 1299, 1304).

The serum of convalescents and of persons long recovered will agglutinate the spirochætes in cultures, and will protect guinea-pigs against infection.　In the writer's experience, 0·5 c.c. of serum and 1 c.c. of culture injected intraperitoneally to guinea-pigs failed to produce even a febrile reaction, whereas 1 c.c. of culture alone produced a rapidly fatal infection.
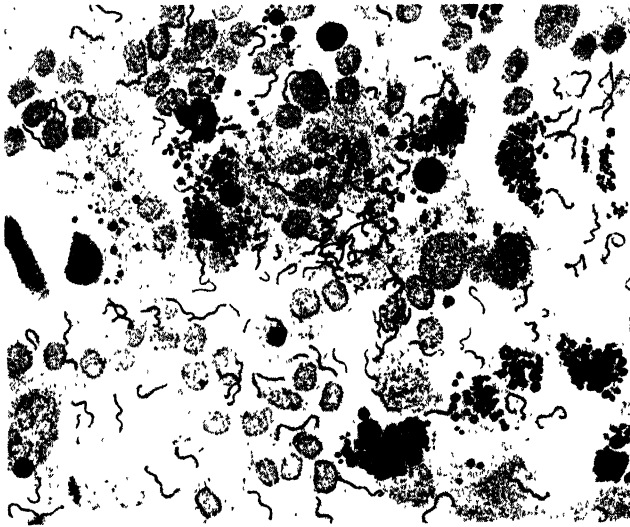
Fig. 560.--*Leptospira icterohæmorrhagiæ* in Section of Suprarenal Gland of Experimentally Infected Guinea-Pig (Silver Nitrate Staining). (× *ca*. 1,000). (After Martin and Pettit, 1919.)

Uhlenhuth and Fromme (1919) noted the presence of antibodies in the blood of a case which had recovered twenty-two years before.

Blanchard, Lefrou and Laigret (1922, 1923) have noted a disease in the Congo which closely resembles Weil's disease.　An outbreak occurred in a hospital, and they had reason to suspect that the vector was the bed bug. Experiments conducted on guinea-pigs, which were very susceptible to the organism and responded as they do towards *L. icterohæmorrhagiæ*, showed that bed bugs were able to transmit the infection thirty-eight days after feeding on an infected animal.　The authors, nevertheless, regard the organism as identical with that of Weil's disease.　Bonne (1924), working

with *L. icterohæmorrhagiæ* in Paris, has found that it will survive for at least two days in bugs, as proved by inoculation of guinea-pigs. He failed to transmit the organism by the bites.

Japanese Seven-Day Fever.—Ido, Ito and Wani (1918) isolated from a case of Japanese seven-day or autumnal fever an organism which, according to them, is morphologically very similar to *Leptospira icterohæmorrhagiæ*. By various serological tests they showed that it differed from the form seen in Weil's disease, while guinea-pigs were not so seriously affected. They named the organism *Spirochæta hebdomadis*. It was discovered that the field vole, *Microtus montebelli*, was a natural reservoir of the virus. Zuelzer and Oba (1923) find that *Leptospira hebdomadis* differs from *L. icterohæmorrhagiæ* in certain details. It is more refractile, thicker, and more loosely wound. Noguchi (1925), who gives its measurements as 10 to 30 microns, also states that it is larger and coarser. Vervoort (1923) has isolated by culture and inoculation of guinea-pigs a leptospira which he calls *L. pyrogenes* from cases of a febrile disease in Sumatra which resembles dengue. In certain patients there is jaundice. The relation of this leptospira to *L. hebdomadis* and those isolated in other diseases has not been determined.

Yellow Fever.—This is a disease of South and Central America and West Africa which in many respects resembles severe forms of Weil's disease. The symptoms, however, are more severe, while hæmorrhages into the stomach and intestine give rise to the characteristic black vomit and melæna. There is a very high mortality rate. Schaudinn (1904) stated that he considered it not improbable that yellow fever would be found to be a spirochætal disease. Stimson (1907) discovered an organism in the kidney of a case of yellow fever after staining by Levaditi's method. On account of its characteristic structure, he doubted if the organism was a true spirochæte, and named it provisionally (? *Spirochæta*) *interrogans*. Noguchi (1919), by inoculations of guinea-pigs with the blood of yellow fever patients in Guayaquil, isolated an organism which was morphologically similar to *Leptospira icterohæmorrhagiæ*. The same organism was isolated directly from cases in serum saline media, and has been the subject of an exhaustive series of investigations by Noguchi (1919–20). It produces in guinea-pigs a condition closely resembling that arising from inoculations with the spirochæte of Weil's disease. Animals which had recovered from an infection with Weil's disease virus were, however, inoculable with the new organism, while the serum of yellow fever convalescents protected guinea-pigs from infections with this organism, but not *L. icterohæmorrhagiæ*. Noguchi (1919a) named the organism isolated from yellow fever cases *Leptospira icteroides*, though he admits (1921) that it is indistinguishable from the one described by Stimson. It is evident from Stimson's figures that the organism he discovered is morphologically iden-

tical with the one isolated by Noguchi, so that the correct name for the yellow fever organism would appear to be *Leptospira interrogans* (Stimson, 1907). The serum of yellow fever convalescents and that of horses and rabbits inoculated repeatedly with cultures of both *L. icterohæmorrhagiæ* and *L. interrogans* were tested against various strains. These sera agglutinated the organisms *in vitro*, gave Pfeiffer's agglutination in the peritoneal cavity of guinea-pigs, and protected these animals against infection. These reactions only occurred with the particular organism which had been used to prepare the immune sera, are thus quite specific, and afford the chief means of distinguishing the two spirochætes. Noguchi (1925) gives the measurements of *L. interrogans* as 4 to 14 microns, and notes that it is somewhat thinner than *L. icterohæmorrhagiæ*.

The distribution of the organism in the tissues of yellow fever cases and in experimental animals corresponds with that in Weil's disease, while the lesions produced in guinea-pigs are very similar to those caused by *L. icterohæmorrhagiæ*. Noguchi and Kligler (1920, 1921) have investigated yellow fever in Mexico and Peru, and have confirmed the earlier work of Noguchi in Guayaquil. Perez Grovas (1921) and Le Blanc (quoted by Noguchi, 1921) have also isolated the organism from yellow fever cases in Vera Cruz, while Noguchi *et al.* (1924) have isolated the organism from cases in Brazil, and proved its identity with the various strains previously obtained in Ecuador, Mexico, and Peru. In spite of criticism by Guiteras and certain discrepancies between the findings of Noguchi and those of the American Commission of 1901, there seems to be very little doubt that *L. interrogans* is the causative agent of yellow fever, as pointed out by Noguchi (1922, 1925). The organism has not yet been isolated from cases of West African " yellow fever."

FIG. 561.—*Aëdes argenteus (Stegomyia fasciata)* (♀), THE TRANSMITTER OF YELLOW FEVER (× 6). (FROM BYAM AND ARCHIBALD'S *Practice of Medicine in the Tropics*.)

L. interrogans is inoculable to guinea-pigs, monkeys, and young dogs, and gives rise to acute infections characterized by jaundice associated with hæmorrhages into the lungs and intestinal mucosa, nephritis, and fatty degeneration of the liver. Like *L. icterohæmorrhagiæ*, it is readily culti-vated. The cultural forms are capable of passing through Berkefeld filters, as demonstrated by Noguchi (1919) and Dieterich (1924).

Though both Beauperthuy and Finlay arrived at the conclusion that yellow fever was transmitted by mosquitoes, evidently *Aëdes argenteus* (*Stegomyia fasciata*), it was the American Commission, consisting of Reed, Carrol and Agramonte (1901), at Quemandos who definitely proved that this mosquito was the carrier (Fig. 561). The work of the Americans was confirmed and extended by Marchoux, Salimbeni and Simond (1903), working in Rio de Janeiro. It was shown that the blood of man is only infective during the first three days of the illness, and it is only during this period that mosquitoes can become infected. The mosquito itself does not become infective till after the expiry of at least twelve days from the time of feeding, after which it can remain so for at least eight weeks. In one experiment the French Commission claim to have demon-strated the infectivity of mosquitoes reared from the eggs laid by infected parents, but there would seem to be some doubt about this observation, which has not received confirmation.

It is a remarkable fact that the mechanism of transmission of yellow fever by *A. argenteus* was discovered before the parasite was known, and that the knowledge thus gained has been so successfully applied that the disease has been to a large extent eradicated.

Noguchi (1919a) conducted transmission experiments with *A. argenteus* and guinea-pigs. A large number were made, but positive transmission only occurred in a small number of these. The mosquitoes were fed on yellow fever cases on the first day of the disease or on infected guinea-pigs. The test for infectivity was made by feeding the mosquitoes on guinea-pigs two weeks later. In a certain number of instances the guinea-pigs were infected. The organism was discovered in the mosquitoes by dark-ground illumination, and the emulsion containing the organisms infected guinea-pigs when smeared on the scarified skin. Noguchi (1925) states that Iglesias has also succeeded in transmitting the infection from guinea-pig to guinea-pig by means of *A. argenteus*.

By means of cultures of the organism Noguchi has prepared a high titre serum by repeated inoculation of horses. There is some evidence that this serum is of definite curative value if given early in the disease. A vaccine has also been prepared from cultures, and the evidence so far obtained indicates that on the outbreak of yellow fever in any locality a large part of the population can be protected against infection.

The structures which were seen in the red blood-corpuscles of yellow fever cases by Seidelin, and named by him *Paraplasma flavigenum*, have been referred to above (p. 1063).

Sand-Fly Fever and Dengue.—Sand-fly fever is a disease of short duration which is conveyed by a species of *Phlebotomus* (Fig. 193). The temperature rises suddenly, remains high for two or three days, and then suddenly falls. Occasionally there is a relapse of a mild type a few days later. Dengue in many respects resembles sand-fly fever, but the febrile period is longer and relapse is more common. A rash is a characteristic feature of the condition, while the transmitting agent is the mosquito (*Culex, Aëdes.* Sand-fly fever occurs in all the countries round the Mediterranean and extends into India. Dengue occurs typically in Australia, but is recorded from India, Africa, and other places. In the Mediterranean region, according to French observers, it is also present, though in many records it appears that the disease described as dengue is the same as sand-fly fever. It is possible that the disease which produces the typical clinical picture of sand-fly fever may occur in other forms which resemble true dengue. Whittingham (1921) succeeded in cultivating a spirochæte from six cases of sand-fly fever in Malta. The blood was taken on the first day of the disease and inoculated to tubes of rabbit blood-agar medium; the organisms became visible in the medium on the fifth or sixth day. Structurally, it closely resembled the *Leptospira* of Weil's disease. It would seem probable that it is the causative organism of sand-fly fever, but this has not yet been definitely established. Couvy (1921), working at Beyrouth, stated that he had seen exceedingly fine spirochætes in the blood of five or six cases of a disease which he calls dengue which were examined just before the onset of fever. This observer (1922) gives a further account of his experiments. He has inoculated the organism to rabbits, and has also infected these animals by inoculating crushed-up *Phlebotomus*. He concludes the disease is carried by *Phlebotomus*, but nevertheless regards it as dengue because of the frequency of relapse and the occurrence of a rash. Whether it is actually dengue or a more severe form of sand-fly fever future investigations alone will show. De Faria (1924) claims to have seen a leptospira in a case of dengue, and proposes the name *Leptospira couvyi* for the organism. Carbo-Noboa (1924), working in Guayaquil, states that he infected guinea-pigs with a leptospira by inoculating them with the blood and cerebro-spinal fluid of cases of dengue. The organism was seen by dark-field examination in the blood and cerebro-spinal fluid of the cases and in the blood and urine of the guinea-pigs. The strain can be maintained in guinea-pigs and in culture media. The name *L. asthenoalgiæ* is proposed for the organism. Knowles and Das Gupta

(1924), during an epidemic of dengue in Calcutta, investigated a number of cases, but obtained no evidence of the presence of leptospira in these.

Blackwater Fever.—Blanchard and Lefrou (1922, 1923) state that by a triple centrifugalization of the blood of three cases of blackwater in the Belgian Congo they have isolated a spirochæte which resembles a leptospira. The organism was inoculable to guinea-pigs, and in these animals gave rise to hæmaturia and hæmorrhages in the tissues. Jaundice, which is such a pronounced feature of guinea-pigs infected with *Leptospira icterohæmorrhagiæ*, was not noted. It is concluded that hæmoglobinuria is a symptom of several diseases, and they name the organism *Spirochæta biliohæmoglobinuriæ*. Schüffner (1918) described a spirochæte from cases of hæmoglobinuric fever in Sumatra, and suggested the name *S. icterohæmoglobinuriæ*. The exact significance of these organisms has not yet been determined, for J. G. Thomson (1923) in Rhodesia and Low and Duncan (1923) in London failed to discover leptospira in cases of blackwater fever, though they followed the technique used by Blanchard and Lefrou.

In Sumatra observers, particularly Vervoort (1923), have isolated leptospira from diseases of various types. These include simple fevers of one to five days' duration with no jaundice, more severe fevers with jaundice and resembling Weil's disease, and, finally, cases with jaundice and hæmoglobinuria resembling blackwater fever. The organism isolated from the mild cases is morphologically identical with that of Weil's disease, and may produce in guinea-pigs symptoms as severe as those obtained from the more serious diseases. Vervoort (1923) proposes the name *L. pyrogenes* for the organism isolated from the fevers of short duration. Bonne (1924*a*) has used the name *S. febrilis* for the same organism. Many more investigations will have to be carried out before the true relationship to disease of the various forms isolated from man, animals, and water is properly understood.

PARASITIC SPIRILLA.

SPIRILLUM OF RAT-BITE FEVER.—Rat-bite fever, or sodoku, is a disease which follows the bite of rats, and is due to infection with a small spiral organism belonging to the genus *Spirillum*, which occurs naturally in the mouth of these rodents. It is of a very chronic nature, and irregular attacks of fever, lasting several days, may recur during the course of many months. At each attack the site of the original wound becomes red and swollen, the glands draining the area enlarge, and an eruption appears on the skin in the form of papules. The organism, which was first seen in the blood by Futaki, Takaki, Taniguchi and Osumi (1916), was named by them (1917) *Spirochæta morsus muris*. Dujarric de la Rivière (1918) objected to this name, as it did not conform to the rules of nomenclature, and proposed the name *Spirochæta japonica*.

The organism is a comparatively broad spiral with a length of 2 to 5 microns (Fig. 562). Occasionally, as Robertson (1924) notes, longer forms 9 to 10 microns in length occur. The number of coils varies from two to four, or even eight or nine in the longest forms. As seen by dark-ground illumination, the spirillum is exceedingly active, and has a perfectly rigid body. One, two, or more flagella may be detected at each end. Reproduction takes place by transverse division at the middle of
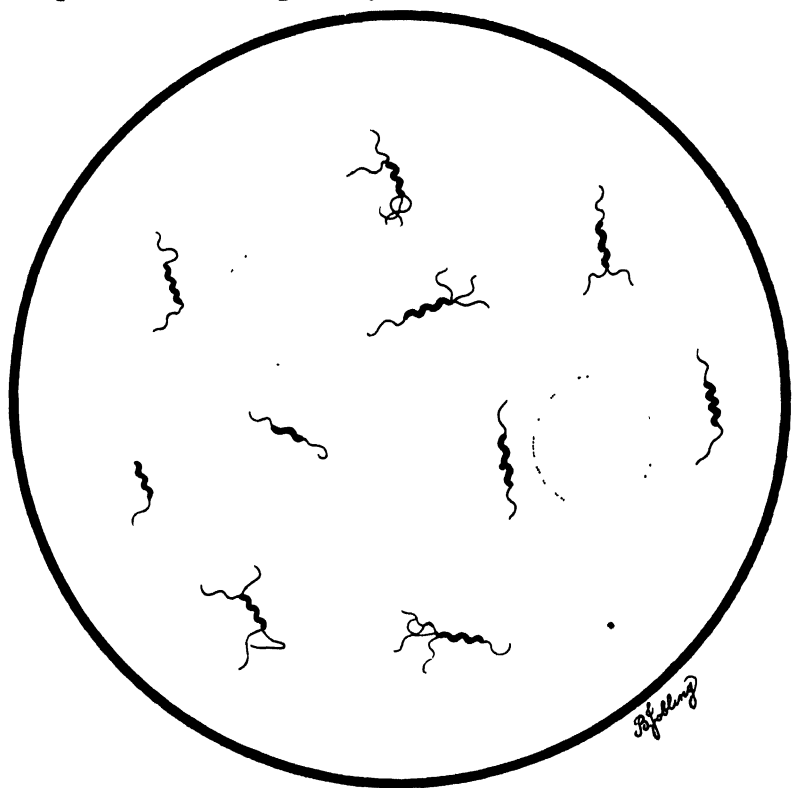
FIG. 562.—*Spirillum minus* OF RAT-BITE FEVER, AS SEEN IN A BLOOD-FILM OF A MOUSE (×2,000). (ORIGINAL.)

The film was subjected to prolonged staining with Leishman stain in order to stain the flagella, which are easily seen by dark-ground illumination of the living organisms. The dotted rings represent red blood-corpuscles.

the body. The organism is readily inoculable to monkeys, mice, rats, guinea-pigs and rabbits. Rats are able to convey infection to guinea-pigs by their bites, an observation of Ishiwara and his co-workers (1917), who state that it had first been made by Ogata. Salimbeni, Kermorgant and Garcin (1925) have noted that infected guinea-pigs may give birth to infected young. Row (1917, 1918) discovered a similar organism in cases of the disease in India, and owing to the fact that he considered it smaller than

that described by the Japanese workers, and that it had no terminal flagella, he (1922) suggested for it the name *Spirochœta petit*. From the work of Parmanand (1923), who demonstrated flagella, there appears to be little, if any, difference between it and the type found in Japan and elsewhere.

An organism which the writer (1906) discovered in mice at the Pasteur Institute in Paris appears to be indistinguishable from the form seen in rat-bite fever. The writer gave animals known to be harbouring the organism to Breinl and Kinghorn (1906), who, a short time before the writer's suggested name *Spirochœta muris* was published, named it *S. laverani*. The same organism had previously been seen by Borrel (1905) in cancer tissues of mice, while it is probable that the form seen by Vandyke Carter (1887) in Indian rats, and named by him *Spirillum minor*, is also identical with it. The same remark applies to *Spirochœta muris* var. *virginiana* MacNeal, 1907, of the rat in America and *S. muris* var. *galatziana* Mezincescu, 1909, of rats in Roumania. Coles (1918) saw the same organism in the blood of an English rat. A similar form was found by Carson in a rat (*Crycetomys gambianus*), which had been inoculated from a West African dog harbouring trypanosomes. Yorke and Macfie (1921), who described it, were inclined to think the organism came from the dog, but subsequently Leeson and Abbott (1924) found it in nine uninoculated rats.

There seems to be little doubt that the organism of rat-bite fever is not a true spirochæte, as Zuelzer (1921) has pointed out. It is more of the nature of a spirillum, with rigid body and terminal flagella, and, unlike a spirochæte, stains very readily with ordinary bacterial stains.

As regards the correct name of the organism, there is much difference of opinion. There is little doubt that the organism which occurs in the human disease is the same as that in rats, and it is highly probable that the various forms found in rodents are all identical with the one originally described by Vandyke Carter from an Indian rat. In this case the correct name for the organism, as Robertson (1924) points out, would be *Spirillum minus* (Carter, 1887).

Futaki, Takaki, Taniguchi and Osumi (1917) stated that they had obtained a culture of the organism. The medium employed was one devised by Shimamine. Nucleinate of soda (0·5 to 0·75 gram) is dissolved by shaking in 100 c.c. of horse serum. and a current of anhydrous carbon dioxide passed through for three or four minutes till the solution is transparent. The tubes are heated to 60° C. for an hour on three consecutive days, and to 65° C. for half an hour on the fourth day. The medium, which has a liquid and a solid part, is then ready for use. After inoculation the tubes are kept at 37° C. for two weeks, when as many as six to ten parasites can be found in each field. Sub-cultures were not obtained, nor were the cultural forms inoculable to animals. In addition to the short organisms

like those which they found in the blood, the cultures were said to contain much longer forms up to 19 microns in length. Many observers have failed to obtain cultures on this and other media, but recently Onorato (1923) claims to have succeeded with Shimamine's medium, as well as with a hæmolysed rabbit's blood medium. The cultures, which were obtained from the blood of human cases, as also the blood and organs of inoculated animals, were maintained by subculture, and were virulent for animals.

Joekes (1925) has grown the organism from the blood of guinea-pigs. To a tube containing a slope of Löffer's inspissated horse serum are added 10 c.c. of a mixture of peptone 1 gm., N. phosphoric acid solution 3 c.c., and distilled water 900 c.c. (pH=7·2). Incubation is at 37° C., and subcultures can be made in the same medium or in a 1 per cent. glucose broth.

Blake (1916) stated that he had isolated from rat-bite fever cases an organism of the nature of a streptothrix, but it evidently had no etiological connection with this disease.

A very similar disease following the bite of cats (cat-bite fever), weasels and squirrels has been described, and Yamada (1917) and others have isolated an organism indistinguishable from *S. morsus muris* from cats. Mooser (1925) has seen a case of sod?ku resulting from the bite of a cat, and has shown that cats and dogs can be infected with the organism found in rats.

Rat-bite fever is very readily controlled by intravenous injections of salvarsan and allied products, which bring about a rapid disappearance of the organisms from the blood and suppression of all symptoms.

OTHER SPIRILLA OF MAN.—Other organisms which have rigid bodies and terminal flagella have been described from the intestinal tract of man and animals. It is possible that *Spirochæte eurygyrata* and some other forms which have been regarded as spirochætes should be included amongst the spirilla. Occasionally such forms have been found in the blood. Pons (1923, 1924) isolated by culture from the blood of a patient suffering from a relapsing type of fever in Saigon an organism to which he gave the name *Spirochæta sinensis*. It is readily maintained by culture in blood media, and varies in length from 3 to 100 microns. The short forms, 3 to 8 microns in length, have two to three loose spirals, and are very motile; the intermediate forms, 15 to 25 microns in length, have four to six spirals, and are also progressively motile, while the long forms are not progressively motile, and show only undulatory movements. In old cultures only coccoid forms can be found, yet sub-culture will yield typical spiral organisms which appear to have rigid bodies. It is very pathogenic to guinea-pigs, rabbits, and monkeys, from the blood of which it can be recovered by culture. This organism seems to be of the nature of a spirillum rather than a spirochæte, and though in preparations it is said to have the typical spiral structure, no satisfactory explanation of the very long forms has been given.

PART IV

METHODS OF INVESTIGATION AND RULES OF NOMENCLATURE

.

METHODS OF INVESTIGATION

I. PROTOZOA.

In this section it is not proposed to give an exhaustive account of the many methods which have been elaborated for the study of Protozoa. The description will be limited to a consideration of certain general principles, and some of those methods which are commonly employed and can be carried out in any laboratory by workers who have already acquired the habit of using the microscope and are acquainted with the ordinary laboratory technique.

The Protozoa do not differ from other animals in that observations on the living organism are essential for a proper understanding of their morphology, physiology, and life-history. It is only by studying them alive that locomotion, reproduction, syngamy, nutrition, and many other functions can be followed. Furthermore, unless the structure of the living organism is known, there will be no means of controlling the changes which may be brought about by the process of fixation and staining.

The study of free-living Protozoa during life is a relatively simple matter, as they can be observed in the media in which they naturally occur. In the case of parasitic forms, there is the great difficulty that directly they are removed from their normal habitat, even if kept in the natural fluids of the host and at their normal temperature, they quickly degenerate. Nevertheless, by the examination of relays of parasites at different stages of development it may be possible to follow the entire life-cycle in the living condition, though not in any single individual.

For a detailed study of the minute anatomy carefully fixed and stained specimens are essential. In the case of parasitic forms, not only must the organisms themselves be thus prepared, but also the tissues of the host. The latter must be fixed with as little disturbance as possible, and examined in serial sections in order to throw light on the true relation of the parasite to the host.

It cannot be too forcibly emphasized that very fallacious pictures of the relation of parasites to cells are obtained by merely smearing tissues on slides. Such a procedure invariably causes the rupture of many cells, the disarrangement of the parts, and the transference of parasites to places in which they do not naturally occur.

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In the case of certain parasites, observations on the living organisms have been facilitated by the discovery of culture media in which they can be maintained alive for many generations outside the body of the host.

For the complete study of the life-history of any parasite it is necessary to follow an infection from its commencement to its termination, and to note the variations which occur. On this account it is advisable to maintain a strain of the parasite in its natural host. This is accomplished by infecting the animals successively either by feeding them on infective forms of the parasite, injecting them by various routes, or allowing the natural transmitting hosts to bring about the same result.

Many parasites are exceedingly polymorphic, so that single isolated observations are apt to be very misleading. It has frequently happened that different stages of one and the same organism have been described as distinct species. It is only by prolonged observation during the entire course of an infection that the range of variation in form and size of any one species can be ascertained.

Study of Living Organisms.

In the case of parasitic Protozoa observations on the living organisms are made by abstracting them from the tissues or fluids in which they occur, and observing them, preferably in the fluid which is taken with them, between a slide and cover-glass. It may be necessary, in order to avoid pressure, to support the cover-glass—e.g., with a hair—and in cases in which it is necessary to use an oil immersion lens, to seal and fix the edges of the cover-glass with paraffin or, better, Czokor's wax, which serves the double purpose of fixing the cover-glass and preventing evaporation. The wax is made by heating together and mixing in a shallow tin provided with a lid equal weights of beeswax and Venetian turpentine. When cool, it forms a solid mass. The wax is applied with a piece of fairly thick wire bent to form a T or L, the cross-piece being about the width of a slide. The cross-piece is heated, placed on the surface of the wax, and then on the slide. The wax and wire can be kept together in the tin.

If the observations are intended to reveal the natural movements and development of organisms, it is necessary to keep the slide at the temperature of the host. For parasites of cold-blooded animals a warming apparatus is not necessary, but when the host is warm-blooded the slide can be maintained at the requisite temperature by some form of warm stage or by enclosing the whole microscope in a warm chamber. In the case of flagellates, the movements are often so active that details of structure cannot be seen till the organisms have become less motile. Those from warm-blooded animals are less active in the cold, and in order to count

the flagella it may be necessary to reduce the temperature. It has to be remembered, however, that the conditions are then unnatural, and may lead to degenerative changes.

The material in which the organisms occur may be too thick, as is often the case with fæces, or contain too many cells for satisfactory observation. It can be diluted with some suitable fluid, ordinary physiological saline solution (0·9 per cent. sodium chloride in distilled water) being very commonly used. In its place the serum of the host itself can be employed.

Various *intra vitam* stains can be added to the liquid, and these have the property of colouring different parts of the organism or material in its vacuoles without for some time appreciably altering its movements or changing it in any other way. The stains most commonly used are methylene blue and neutral red. They are made up in very dilute solution (1 in 500 to 1 in 10,000). A small quantity can be mixed with the fluid containing the parasite, or a drop of the stain can be allowed to dry on a slide, and the fluid containing the organisms to be examined placed on the same area and covered with a cover-glass. The stains are slowly absorbed by the parasites. Neutral red assumes a bright cherry-red colour in acid and a brown in alkali. On this account it serves as an indicator of the reaction of the substances which it stains. It is largely used for following the process of food digestion in vacuoles.

Another stain which is of value is eosin. It serves as an indicator of the life or death of a cell. Living cytoplasm will not stain, but immediately death has occurred the stain is absorbed. If fæces containing non-motile amœbæ or cysts are mixed with eosin, the living ones which remain unstained can at once be distinguished from those that are dead.

Parasites abstracted from their hosts, and observed as described above, will continue to behave for some time as they would have done if they had not been disturbed. Malarial parasites, for instance, will continue to develop for several hours in what appears to be a perfectly normal manner in a blood preparation kept at the temperature of the body. The production of merozoites can be followed if the observation is commenced on a parasite which is approaching this stage of development. Similarly, the division of trypanosomes can be watched, the multiplication of nuclei in amœbic cysts, and many other developmental processes. On the other hand, all parasites must eventually leave one host in order to gain access to another. In so doing they either escape on to the ground or into water, where, with or without further development, they await the next host, or they enter an invertebrate and are transferred by it to the next vertebrate host. Those parasites which have

a stage which undergoes development outside the host may be directly observed in the living condition. Thus, the oöcysts of coccidia of warm-blooded animals escape in the fæces in an incompletely developed condition, and may be observed to complete their development into sporocysts and sporozoites on the slide. Cysts of amœbæ containing glycogenic or chromatoid bodies may be seen to lose these. Without continuously observing any individual cyst, material containing many cysts can be kept and examined at intervals for developmental changes. In the case of the coccidia this is necessary in order to determine whether the cysts are actually oöcysts or not, and if so, to which genus (e.g., Eimeria or Isospora) they belong. To retard the growth of fungi, material can be spread on charcoal or, in the case of oöcysts of many coccidia, mixed with a 5 per cent. solution of bichromate of potash. In the case of parasites which enter invertebrates, their removal from the body of the vertebrate on to a slide may cause them to commence the invertebrate cycle. Thus the production of gametes from the gametocytes of hæmosporidia, the process of fertilization, and the development of the oökinetes may be followed in the living condition in a single drop of blood between a slide and cover-glass. The further development of the oökinete, which takes place in the tissues of the invertebrate, has not been followed in this manner, though it might be done by dissection of the mosquitoes and observing the parasites in the natural fluids and tissues. Similarly, the early developmental stages of trypanosomes in the invertebrate, especially those of cold-blooded animals, can be observed on the slide in blood preparations made from the vertebrate (see pp. 594 and 603). Another illustration of the value of this method of observation is afforded by Leishmania. If spleen pulp containing L. donovani is placed in a suitable liquid (e.g., simple physiological saline or the liquid in N.N.N. medium), the organisms elongate and become flagellated. By examining the fluid at intervals, the whole development of the leptomonas from the leishmania form can be followed in the course of forty-eight hours. Observations of this kind must always be controlled by a study of the changes which actually take place in the invertebrate, in order to avoid the confusion of what may be degenerative changes with those which form a normal part of the life-cycle.

At any time during the observation of parasites between a slide and cover-glass, the cover-glass can be removed and films made with the material. Thus, in the case of malarial parasites a preparation can be observed till microgamete formation is taking place. By removing the cover-glass and making a film from the blood as described below, permanent preparations of the process, together with that of the development of the macrogamete and actual fertilization, can be obtained.

Observations are usually made by transmitted light, but dark-ground illumination may be of service in the detection of fine filaments such as extrusion filaments of cnidosporidian spores, flagella, and cilia.

Culture Methods.

The maintenance of Protozoa in artificial culture media is an important method of investigation. To be successful, such media must contain the substances which are essential to the natural growth of the organism, and must imitate as nearly as possible the natural environment. For free-living Protozoa, including coprozoic organisms, the necessary conditions are not difficult to attain, but in the case of parasites the preparation of suitable media is beset with greater difficulties. This is more readily accomplished for parasites which live in the fluids of the host. In the case of parasites which are definitely intracellular there is much greater difficulty, and, with the exception of species of *Leishmania* and certain trypanosomes (*T. cruzi*), in no instance has the satisfactory culture of such an organism been obtained.

If a culture method is successful, it will be found that the organisms reproduce and increase in numbers for a varying time till the medium is exhausted or has become charged with substances which prevent continued growth. The transference of some of the parasites to fresh medium will be followed by renewed multiplication. By repeated sub-culture it should be possible to maintain the organisms indefinitely.

Certain organisms, as, for example, trypanosomes, leishmania, and the allied insect flagellates, will readily grow in this manner in N.N.N. and other blood media. It is essential that bacteria be absent from the cultures, as they quickly generate substances which kill the Protozoa. In the case of intestinal organisms (amœbæ, flagellates, ciliates) which grow readily in various egg media, the presence of bacteria appears to be a necessity, as the Protozoa feed upon them. Similarly, coprozoic organisms, whether grown in liquid media or on the surface of agar plates, require bacteria as food. When cultivating those organisms which will not grow in the presence of bacteria, the greatest care has to be exercised in the handling of the material to prevent bacterial contamination. If a culture is to be started from the blood during life of the host, the blood may be obtained from the finger, ear, or vein, the skin being carefully sterilized before puncture, which is made with a sterile needle or syringe, the blood being then transferred to the sterile medium with all precautions. Similarly, sterile syringes are used for the puncture of the liver, spleen, or other organs. After death of the host, blood can be obtained from the heart or other organ, the same care being taken to sterilize the surface through which puncture

is made. Even then bacteria may appear in the culture, as these organisms often spread to the blood and tissues from the intestine after death.

When a culture is commenced, a varying quantity of material containing the organism is introduced into the culture medium, which is then kept at the requisite temperature. It may happen in such cases that though it was supposed that only one was introduced, another was also present, and that a culture of two or even more organisms is obtained. Though in many cases it is safe to assume that only a single one is present, for very accurate work it may be necessary to have absolute assurance of this fact. This can only be accomplished by commencing a culture from a single individual. In the case of comparatively large organisms such as ciliates this is a simple matter, but with small ones it is much more difficult, though even then it can be done. The plate method, which is so largely used for the isolation of pure strains of bacteria, has been applied to the Protozoa. If such a pure strain can be obtained, it becomes a matter of certainty that all the various forms which appear in the culture belong to the one species.

When once a satisfactory medium for the culture of any organism has been devised, it may be employed for diagnostic purposes. Material which does not contain an organism in sufficient numbers for it to be discovered by microscopic examination may be inoculated into the medium. If the organism is present it may multiply, so that in a few days the number present is sufficient for its detection. This method has been largely employed for the diagnosis of leishmania and trypanosome infections, and is now being used for the intestinal Protozoa.

Cultivation of Intestinal Protozoa of Vertebrates.

The only Protozoa which have been satisfactorily cultivated are those which live in the lumen of the intestine, including the pathogenic amœbæ and ciliates which invade the intestinal wall. It is not definitely known, however, that these amœbæ and ciliates cannot live and multiply in the lumen of the intestine. Those Protozoa which are true tissue parasites, like the coccidia, and also species of *Giardia*, which inhabit the lumen of the intestine only, will not grow in any artificial medium so far discovered. Those which have been cultivated are the various intestinal amœbæ, flagellates such as *Trichomonas, Chilomastix, Embadomonas, Tricercomonas,* and the ciliate *Balantidium coli.*

Boeck's Locke-Egg-Serum Medium (L.E.S. Medium).—This medium is prepared in the following manner:

1. Four eggs are washed, brushed with alcohol, and broken into a sterile flask containing glass beads. Fifty cubic centimetres of Locke's physiological solution are then added, and solution effected by shaking.

2. Test-tubes are then filled with a sufficient quantity to produce slants about 1 to 1½ inches in length upon coagulation by heat.

3. These tubes are now slanted in an inspissator, and heated at 70° C. till the egg mixture has solidified. They are then transferred to the autoclave, and sterilized for twenty minutes at a pressure of 15 pounds.

4. To each tube is now added a mixture of eight parts of sterile Locke's solution and one part of inactivated human serum till the liquid reaches a height of 1 centimetre above the egg slant.

The Locke's solution, which is sterilized in the autoclave or steamer, has the following composition:

Distilled water	1,000 c.c.
NaCl	9·0 grams.
CaCl	0·2 ,,
KCl	0·4 ,,
NaHCO₃	0·2 ,,
Glucose	2·5 ,,

The human serum must be sterile, as determined by the inoculation of bacterial media. If there is doubt as to its sterility, it can be diluted with two or three parts of Locke's solution, and filtered through Berkefeld filters (No. N) once or twice to remove the bacteria. When sterile, it can be diluted with the necessary amount of Locke's solution to bring the dilution to 1 in 8, as required for the medium. When the medium is ready for use, it will consist of a slope of coagulated egg in the diluted serum.

A modification of the L.E.S. medium is the L.E.A. medium of Boeck and Drbohlav. It differs from the L.E.S. medium in that the diluted serum is replaced by a 1 per cent. solution of crystallized egg albumen in Locke's solution sterilized by filtration through a Berkefeld filter. The albumen solution may be prepared by using the white of one egg in 1,000 c.c. of Locke's solution, instead of the crystallized albumen. Another modification consists in replacing the egg slope by an ordinary blood-agar slope, such as that of N.N.N. medium. The reaction of these various media should be pH=7·2 to 7·8. When they have been prepared as directed, the reaction will usually be found to be at this level.

This medium, or modifications of it, has been used by Boeck and Drbohlav for the cultivation of *Entamœba histolytica, E. coli, E. gingivalis, E. aulastomi,* and the flagellates *Tricercomonas intestinalis, Chilomastix mesnili, Trichomonas hominis, C. gallinarum, T. gallinarum,* and *T. sanguisugæ.* Thomson, J. G., and Robertson have cultivated all the intestinal amœbæ of man, as also the human flagellates just mentioned, while Dobell (1926) has cultivated *E. histolytica, E. gingivalis* and four amœbæ, and a *Trichomonas* and *Tricercomonas* of monkeys. The medium will undoubtedly be found to be successful for many other intestinal Protozoa. The cultures are commenced by introducing into

the medium a small quantity of fæces or intestinal mucus in which the organisms are present. The material should be placed with a pipette at the bottom of the tube. The tubes are then incubated in the vertical position in an incubator at 37° C. in the case of parasites of warm-blooded animals. From time to time material for examination can be removed from the bottom of the tube by means of a pipette. In the case of amœbæ it is advisable to scrape the surface of the egg slope with the end of the pipette to remove amœbæ before sucking up the material. As the movements of amœbæ are of such importance for their identification, the slides should be examined on the warm stage or in a warm microscope chamber. For identification of flagellates this is not so necessary. Multiplication takes place for two or three days, after which the bacteria outgrow the Protozoa and bring about their death. Subcultures should be made every two days by sucking up some of the organisms with a pipette and transferring them to a fresh tube of medium. If a small quantity of blood be added to the medium, or if the blood-agar modification be used, it will be seen that *E. histolytica* ingests the red blood-corpuscles.

Drbohlav (1925a) has come to the conclusion that the preparation of the medium can be simplified and improved. For the liquid part of the medium he uses Ringer's solution, to each litre of which 5 grams of monopotassium phosphate is added as a buffer, KOH or NaOH solution in quantity sufficient to bring the pH to 7·4, and the white of an egg which has been removed from the shell with all precautions against bacterial contamination. Filtration of the mixture is not carried out, but the heavy material is allowed to separate by sedimentation. For the solid agar slopes three modifications can be used:

1. Ringer's solution with buffer (pH=7·4)   ..   ..   1 litre.
    Agar   ..   ..   14 grams.
2. Ringer's solution with buffer (pH=7·4)   ..   ..   1 litre.
    Agar   ..   ..   14 grams.
    Starch   10   ,,
3. Blood agar slopes (N.N.N. medium) heated for thirty minutes at 100° C.

In all these media the amœbæ grow well, and especially in the second and third may survive for two or three weeks, probably because the bacterial growth is checked. The addition of 1 per cent. dextrin to the liquid portion has the property of suppressing the growth of *Blastocystis*, which frequently multiplies rapidly and exterminates the amœbæ. One or two subcultures in the dextrin medium will rid the culture of this organism, after which a return to dextrin-free medium can be made.

Hogue's Egg Medium.—This is a liquid medium prepared by mixing a whole egg with 200 c.c. of Locke's solution. The mixture, constantly stirred, is steamed for fifteen minutes over the boiling water-bath and then

filtered through cotton-wool. The filtrate is then distributed in quantities of 5 c.c. to test-tubes, which are sterilized in the autoclave at 120° C. for twenty minutes. The medium, which consists of a clear liquid and a granular sediment, has been used for the cultivation of *Embadomonas intestinalis* and *Trichomonas hominis* by Hogue. The writer has used it for the cultivation of *Embadomonas* from man, guinea-pig, rat, tortoise, and frog, and for *Trichomonas* from various sources. In the case of parasites of warm-blooded hosts the temperature of incubation is about 37° C., while for those of cold-blooded hosts the ordinary laboratory temperature will suffice. On this medium Hogue obtained cultures of pure strains of *T. hominis* commenced from a single individual.

Serum Media.—Intestinal flagellates have been successfully grown in various serum media consisting of physiological saline solutions to which varying quantities of serum have been added. The best results appear to have been obtained by Ohira and Noguchi (see p. 652) in a medium consisting of equal parts of ascitic fluid and Locke's solution. In this they successfully cultivated and maintained in subculture the *Trichomonas* of the human mouth. Subculture every two or three days was necessary. In the same medium *Entamœba gingivalis* lived and multiplied for ten days.

Barret and Smith have successfully cultivated an amoeba (*Entamœba barreti*) of the tortoise in a mixture of one part of human serum and nine parts of 0·5 per cent. solution of sodium chloride (see p. 207). *Blastocystis* also grows readily, and may multiply to such an extent that the amoebæ are prevented from reproducing. The amoebæ were maintained in this medium at room temperature, or even in the ice-box (10° to 15° C.), for many months, during which subculture was made every two or three days or every week according as the temperature of incubation was high or low. Barret and Yarbrough have cultivated *Balantidium coli* in a similar manner (see p. 1207).

Semi-Solid Blood-Agar Medium.—To 270 c.c. of 0·85 per cent. sodium chloride solution (pH=7·6) are added 30 c.c. of ordinary 2 per cent. bacteriological nutrient agar (pH=7·6). When mixture has taken place, 10 c.c. are placed in each of a series of test-tubes. After autoclaving at 120° C., the tubes are cooled to 50° C., and into each tube are allowed to fall from a rabbit's ear twenty drops of blood. The tubes are not shaken or mixed, and are incubated for twenty-four hours at 37° C. The medium is then ready for use. The blood is obtained from the rabbit's ear by the method described below for the preparation of N.N.N. medium. In this medium, in addition to *Leptospira, Trypanosoma lewisi, Leishmania,* and *Leptomonas* of the flea, certain intestinal flagellates of man and animals have been cultivated, such as *Embadomonas* and *Trichomonas.*

As in the case of other media, if used for the cultivation of intestinal flagellates in the presence of bacteria, it is necessary to subculture every two or three days.

Cultivation of Protozoa from Blood and Tissues of Vertebrates.

The cultivation of Protozoa from the blood or tissues of vertebrates only succeeds if bacteria are kept from the cultures. Though certain forms, such as *Leishmania*, may survive for a few days in the presence of bacteria, they are ultimately killed, probably as a result of the bacterial toxins. As noted above, the material, whether blood or tissues, must be obtained under strict aseptic conditions. The parasites grown are the various blood parasites belonging to the genera *Trypanosoma, Trypanoplasma, Leishmania, Plasmodium,* and *Babesia.* The last two, being inhabitants of the red blood-corpuscles, require these cells in the medium. As all these parasites have both vertebrate and invertebrate hosts, it follows that the cultures may consist of the forms which belong to the vertebrate cycle or those which belong to the invertebrate. As regards *Plasmodium* and *Babesia,* the cultures are incubated at 37° to 40° C., and the forms which appear are those which occur in the vertebrate. In the case of the flagellates from warm-blooded hosts, it is usually found that the best growth is obtained at temperatures well below that of the vertebrate, and the forms which appear in the cultures represent the invertebrate cycle. Cultures of trypanosomes from cold-blooded hosts, though maintained at the temperature of the vertebrate, nevertheless contain forms characteristic of the invertebrate cycle (see p. 606). On the other hand, the pathogenic trypanosomes of vertebrates are difficult to cultivate. They grow only at a temperature approaching that of the host from which they are taken, and the forms which appear in the cultures retain the trypanosome structure, the crithidia forms characteristic of the invertebrate cycle not appearing.

CULTIVATION OF FLAGELLATES.—Blood Agar (N.N.N. Medium).—Novy and MacNeal were the first to cultivate trypanosomes in the condensation liquid of a solid blood-agar medium consisting of nutrient agar to which fresh rabbit's blood was added. The medium was simplified by Nicolle, who showed that simple agar could replace the nutrient agar. The medium—Nicolle's modification of Novy and MacNeal's medium—is generally known as the N.N.N. medium. It is prepared by adding rabbit's blood to melted agar in test-tubes, mixing rapidly, and allowing the mixture to solidify in the sloped position. A solid blood-agar slope is obtained. The tubes are incubated at 37° C. for twenty-four hours in the vertical position to prove sterility and to encourage a liquid to

separate or condense in the lower part of the tube. It is in this liquid that growth occurs. The agar is made by heating the following ingredients together in a flask:

Water	..	..	..	..	900 c.c.
Agar	..	..	..	..	14 grams.
NaCl	..	..	..	..	6 ,,

The mixture while hot is poured into test-tubes to the height of 3 to 4 centimetres. The tubes are then plugged and sterilized in the autoclave. When they have cooled to 50° C. and are still liquid, a quantity of sterile rabbit's blood varying from 2 to 3 c.c., or even more in special cases, is added to each tube. The tubes are rapidly revolved to bring about mixture without forming bubbles and placed in the sloped position, so that the agar solidifies leaving one side of the tube completely free.

Various methods for adding the blood are used. Many workers draw the blood directly from the heart of the rabbit by puncture through the thoracic wall with a large sterile syringe. The blood is immediately transferred to the tubes before any clotting in the syringe has occurred. Other observers remove the blood from an animal in a similar manner, and transfer it to a sterile bottle con-

FIG. 563.—BOTTLE WHICH MAY BE USED FOR OBTAINING BLOOD FROM THE HEART OR VEIN OF ANIMALS.

taining beads. The blood is defibrinated by shaking, and can be placed in the tubes by means of a pipette. Animals tolerate the operation very well, and can be used repeatedly.

Another method which can with advantage be used when chances of bacterial contamination from the air are great is to bleed the animal to death under an anæsthetic. A bottle containing glass beads, and fitted with a cork through which pass two glass tubes, is used (Fig. 563). To one tube is attached, by a rubber tube fitted with a clip, a hollow syringe needle of fairly large bore, while to the other is attached, also by a rubber tube, a small glass suction-tube. The glass tube to which the latter is attached should be plugged with cotton-wool. The whole apparatus, the needle being kept in a test-tube, is sterilized. The heart of the chloroformed animal is then exposed with due precaution as regards sterility,

the sterile needle removed from the test-tube and passed into the heart. Suction applied to the suction-tube causes blood to flow into the bottle. When no more blood is available, the rubber tube to which the needle is attached is clipped, and the blood rapidly defibrinated by shaking the bottle. The same apparatus can be used for abstracting blood from the jugular vein of larger animals such as sheep during life.

A very simple method, which gives good results when chances of con-tamination are less than in the tropics, is to allow blood to drop into the tubes from the paraffined ear of the rabbit. The animal is enclosed in a box, the capacity of which can be adjusted by a movable partition (Fig. 564). At one end is an opening fitting the neck, and through which the head projects when the box is closed. The ear is shaved over the
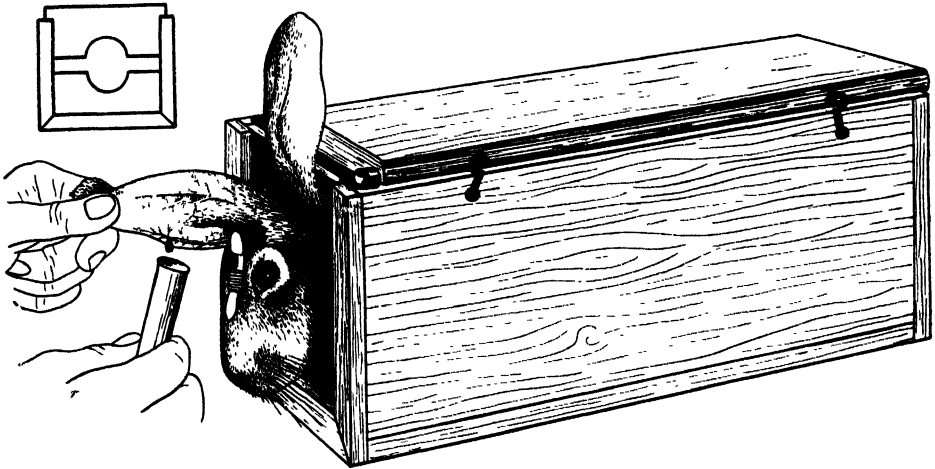
FIG. 564.—Box employed for holding a Rabbit during Operation of Abstraction of Blood from the Marginal Vein of the Ear.

Inset the end of the box, showing method of fixation of neck.

marginal vein, and the skin sterilized with alcoholic iodine solution. When the ear is dry, the area to be used is coated above, below, and on the margin with hot melted paraffin, so that the region of operation is covered by a thin layer through which the vein is still visible. A pressure clip having been placed over the vein on the margin of the ear near its base, an incision is made in the marginal vein with a small sharp knife, and the sterile blood allowed to drop from the margin of the paraffined ear into the tubes. About twenty drops give a sufficient quantity of blood for each tube. It requires at least three persons to carry out the manipulations satisfactorily if blood is not to be wasted. When a batch of tubes has been made and proved to be sterile after incubation, the cotton-wool plugs should be covered with sterile rubber caps to prevent

drying of the medium, which in any case does not preserve its properties for more than a few weeks. Kept in the ice-box it lasts longer.

When it is desired to obtain cultures of a flagellate, a small quantity of blood or tissue containing the organism is introduced into the condensation liquid at the bottom of the tube with a glass pipette or a platinum loop. The tubes are then incubated, usually at 22° to 25° C. A drop of liquid on a sterile platinum loop is removed for examination from time to time. Growth of the organisms takes place not only in the liquid of condensation, but also on the surface of the agar above the liquid. A scraping from this surface will often reveal an enormous number of organisms. The various species of *Leishmania*, the non-pathogenic trypanosomes of warm-blooded hosts such as *Trypanosoma lewisi* of rats and those of birds, and the trypanosomes of cold-blooded animals grow readily on this medium. Subculture into fresh tubes should be made about once every two weeks. Sometimes growth occurs if subculture is not made for six months or more. Nicolle (1925) has pointed out that a strain of *L. donovani* isolated in Tunis has been kept in culture for nearly fourteen years, during which it was subcultured 395 times. A strain of *L. tropica* has been kept for over fourteen years, and has been subcultured 384 times. *L. tarantolæ* has been kept for seven years with 188 subcultures, and a trypanosome of the toad for a similar period with 193 subcultures.

This medium is frequently employed for diagnostic purposes to discover parasites which are present in numbers too small for detection with the microscope. For the diagnosis of leishmania infections in man and experimental animals it is of special value. Similarly, trypanosome infections of birds and cold-blooded animals can be discovered by its means. Infections of geckos with *L. tarantolæ* have been detected in this way. The method has also been applied to insects, such as bed bugs, with the object of determining the length of time *L. donovani* will survive in them.

As noted above (p. 643), Ponselle has cultivated *Trypanoplasma varium* on what is essentially this medium. The agar to the strength of 2 per cent. is dissolved in tap water without any addition of sodium chloride, and with the quantity in each test-tube is mixed an equal quantity of defibrinated rabbit's blood.

Various modifications of N.N.N. medium have been used with success. In the place of rabbit's blood, that of other animals can be employed, while the addition of glucose to the agar may be an advantage. Nöller (1917) and Hoare (1923) have used the following modification for cultivation of the trypanosome of the sheep: Agar 25 grams and glucose 20 grams are dissolved in 1,000 c.c. of broth (pH=7·6). Equal volumes

of agar and defibrinated or undefibrinated horse or rabbit blood are mixed in each tube, which is then sloped.

A further modification introduced by Nöller (1917) is the use of the medium on agar plates. The agar is prepared by dissolving agar 10 grams and glucose 10 grams in 1,000 c.c. of broth. This is placed in test-tubes to a height of 12 to 18 centimetres in each, sterilized, and kept till required. To make a plate, the agar is melted and transferred to a small flask, where it is mixed with an equal volume of defibrinated horse blood. The mixture is immediately poured into a Petri dish, and allowed to solidify. The plates are kept in the inverted position to prevent condensation water falling on the surface, while to avoid drying and contamination, a small quantity of sublimate solution is run into the lid. This has to be renewed from day to day, care being taken to prevent any of it coming into contact with the medium. On these plates Nöller has cultivated many flagellates, including *Leishmania donovani, Trypanosoma theileri,* and *T. melophagium,* trypanosomes of birds, *Leptomonas ctenocephali,* and *L. fasciculata.* The plates are inoculated by streaking them as in bacteriological work. Visible colonies, often branched, are obtained, and in these enormous numbers of the organisms are present.

Noguchi's Serum Medium.—Though the blood and tissue flagellates have been usually cultivated in media containing fresh blood, as described above, many attempts have been made to use blood-free media. Kligler (1924) has shown that *Leishmania tropica* will grow very well in the medium devised by Noguchi for the cultivation of spirochætes of Weil's disease and relapsing fever. Noguchi (1924) has also used it for the cultivation of various species of *Leishmania.* The medium as described by Noguchi and Lindenberg (1925) has the following formula:

Saline solution (0·9 per cent.) .. .. 800 parts.
Fresh rabbit serum .. .. 100 ,,
Nutrient agar (2·0 per cent., pH = 7·2) .. .. .. 100 ,,
Rabbit hæmoglobin solution (made by laking 1 part of
    defibrinated rabbit's blood in 3 parts of distilled water) 10 to 20 parts.

Growth occurs at the top of the medium as a whitish cloud or scum consisting of a mass of organisms. This layer, which will re-form in the course of a week or two, can be removed repeatedly during several months.

Kligler (1925) has used with success the following modification: each tube contains 4·5 c.c. of agar (0·2 per cent. nutrient agar plus 0·1 per cent. dextrose). Just before use 0·5 c.c. of fresh rabbit serum is added to the warmed tube.

Blood-Broth Medium.—In certain cases trypanosomes will grow and multiply in mixtures of blood and ordinary bacteriological broth. The method of growth has been employed chiefly for the cultivation of trypanosomes from the blood of cattle and sheep when they are present in the

blood in small numbers. Blood is taken from a vein by means of the bottle described above (p. 1301). It is defibrinated and distributed in test-tubes, in each of which is a volume of broth equal to that of the blood to be added. The tubes are incubated at 30° C. At the end of a week or ten days, if trypanosomes are present and bacterial contamination has been avoided, the trypanosomes will have multiplied sufficiently to be detected. Subculture cannot be satisfactorily made in the same medium. For this purpose N.N.N. medium, or Nöller's modification of it, must be employed. The flagellates which occur in the cultures tend to be of the crithidia type, an indication that they represent the insect phase of development (see p. 505).

Ponselle's Medium.—This medium is one which Ponselle has used with success for the cultivation of pathogenic trypanosomes. The media described above give good results with non-pathogenic trypanosomes and the various leishmania, but are not satisfactory for those trypanosomes which produce disease, though Novy and MacNeal were able in some cases to cultivate *T. brucei*, and to maintain it in subculture in their original blood-agar medium. Subsequent observers have invariably failed to carry on the cultures which have been commenced in this medium. Ponselle has found the following to give good results:

NaCl	0·3 to 0 8 gram.
Peptone (Witte's)	2 grams.
Gelatin	2 ,,
Normal solution of sodium carbonate	1 c.c.
Distilled water	100 c.c.

All the ingredients must be perfectly pure. They are heated rapidly on a water-bath, and the resulting solution is sterilized in an autoclave at 110° C. for half an hour. When the liquid has cooled to the laboratory temperature, there is added an equal volume of rabbit's serum when medium for a primary culture is required, or an equal volume of de-fibrinated rabbit's blood when the medium is required for subcultures. The mixture, distributed in quantities of 3 c.c. in test-tubes, is then in-activated by keeping it for half an hour at 56° C. As regards the sodium chloride, the quantity required varies with the trypanosome. It is 0·3 for *T. brucei*, 0·6 for *T. pecaudi*, and 0·8 for *T. rhodesiense* and *T dimorphon*.

CULTIVATION OF MALARIAL PARASITES AND PIROPLASMATA.—The cultivation of these organisms differs from that of the blood-flagellates in that they are essentially parasitic in red blood-corpuscles, which must be present in the medium. The outlines of the method for the cultivation of malarial parasites first introduced by Bass and Johns have been noted above (see p. 967). In most cases it consists in keeping the young parasites outside the body for a length of time sufficient for them

to grow and reproduce by schizogony. Occasionally it has been possible to follow the entry of the merozoites into other red cells and to trace their growth into schizonts. More rarely a third generation has been observed. Longer than this it has not been possible to maintain the parasites in cultures, so that the method cannot be compared with those adopted for the blood-flagellates, which can be maintained indefinitely. As in the case of the cultures of flagellates, it is essential that all bacterial contamination be avoided during the various manipulations, which, according to Bass (1914), are as follows:

Blood containing young forms of malarial parasites is taken from a vein with a syringe, from which it is transferred immediately to a defibrinating flask containing 0·1 c.c. of 50 per cent. solution of dextrose for each 10 c.c. of blood. Defibrination is effected by stirring with a glass rod, care being taken to avoid bubble formation. The defibrinated dextrose blood is then transferred to culture-tubes not less than 1·25 centimetres in diameter and 12·5 centimetres in depth. The quantity of blood for each tube may vary in depth from 2·5 to 5 centimetres, which will give a column of serum 1·25 to 2·5 centimetres deep above the cells when they have settled to the bottom of the tube. The column of serum should not be less than 1·25 centimetres, but it may be more than 2·5 centimetres, though there is no advantage in this. The tubes are incubated in the vertical position at a temperature of 40° C. The parasites live and develop in the red blood-corpuscles at the top of the column of deposited cells in a layer which varies in thickness from 0·05 to 0·1 centimetres. All parasites beneath this layer die in from two to twenty hours. The parasites in the thin layer can be examined from time to time by carefully drawing off cells by means of a fine pipette. In this manner it is possible to follow in stained films the growth of the young parasites till they reproduce by schizogony.

If it be desired to obtain more than one generation in the culture, the defibrinated glucose blood must be centrifuged at a speed sufficient to cause the leucocytes to occupy the upper layers of the deposit, so that red cells free from leucocytes can be obtained. The supernatant serum is then transferred to culture-tubes, which are better if they have flat bottoms. The column of serum in each should be from 1·25 to 2·5 centimetres in depth. A pipette is passed into the middle of the deposit in the centrifuge-tube, and red cells and parasites drawn off. These are transferred to the bottom of the culture-tube. By this technique the presence of leucocytes, which devour the merozoites as soon as they escape from the red blood-corpuscles, is avoided. The young parasites will enter red cells and again grow into schizonts. It has only been possible to conduct the parasites through three generations, after which they cease to grow and die.

The above technique has been modified by various observers, but no better results have been obtained.

Row (1917) and Sinton (1922) have devised methods by which the growth of a single generation of parasites can be followed when only a few drops of blood are abstracted. The method is essentially the same as that of Bass. In Row's method the blood is drawn from the finger into a small tube in which it is defibrinated. The small quantity of defibrinated blood is then transferred by means of a pipette to a small, flat-bottomed tube containing serum to which the requisite quantity of dextrose has been added. The small tube is placed in a larger tube (the ordinary bacteriological potato-tube) provided with a constriction on which it rests. Below the constriction the outer tube contains a solution of pyrogallic acid, to which sodium hydrate is added just before the introduction of the small tube. The large tube is then corked tightly with a rubber cork, the pyrogallic acid and sodium hydrate absorbing the oxygen, so that the culture takes place in an oxygen-free atmosphere.

In Sinton's method, all the manipulations are performed in a specially constructed tube about 20 centimetres in length (Fig. 565). It is made from tubing having a bore of 0·4 to 0·5 centimetre. To construct it, one end is drawn out as in an ordinary pipette, and by placing a narrow metal tube over the thin drawn-out portion while it is still soft and pressing upwards there is produced a flat surface, from the centre of which the thin, drawn-out tube arises (A). The tube is now heated a short distance above the flattened surface, and drawn out till it forms a tube about 0·2 centimetre wide (C). There is thus formed a narrow tube which passes into a dilated bulb (B) with its lower surface flattened. At the upper portion of the narrow tube a slight constriction (D) is made, while about 0·4 to 0·5 centimetre above the constriction the tube is again heated, and an indentation made on each side by pressing inwards with the point of an iron nail (E). Three glass beads (F) are then dropped into the upper portion of the tube (G), their progress being checked by the indentations (E). The upper end of the tube is then drawn out and bent,
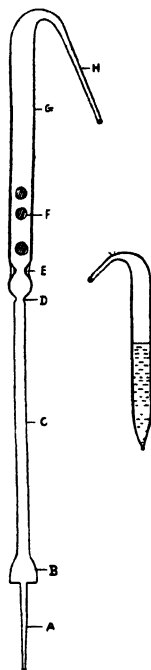
Fig. 565.—Apparatus used by Sinton for Cultivation of Malarial Parasites in Small Quantities of Blood. (After Sinton, 1922.)

The small Wright's capsule contains the ascitic or hydrocele fluid.

as in a Wright's capsule (H).   The upper and lower drawn-out ends are kept sealed, and the whole tube can be sterilized.

When the culture is to be made the tube is opened at both ends, and the upper capillary end is bent at right angles to the plane of the rest of the tube, so that the whole will lie on a table with the open upper end pointing upwards.   The upper end is inserted into an already prepared Wright's capsule containing ascitic or hydrocele fluid to which the requisite quantity of dextrose solution has been added.   The fluid is allowed to enter the tube till the upper section is about a third or half full.   From the carefully sterilized finger of a malarial patient five to ten drops of blood are run into the fluid in the tube.   The dilated part of the lower end of the tube is then gently heated and the narrow tube below it sealed off.   As the air in the dilated part cools, the blood-mixture is drawn further into the tube.   After this the upper end is sealed off.   By shaking the glass beads the blood-mixture is defibrinated.   When this is complete, the tube is swung round rapidly so as to drive the defibrinated blood-mixture through the constriction into the lowermost part of the tube, where it should fill the dilated part and the narrow section above it.   The tube is then heated at the constriction above the column of fluid and sealed off.   The red corpuscles settle to the flat bottom of the dilated lower portion, and the whole tube is incubated in the vertical position.   In order to examine the culture the tube has to be opened.   After removal of cells by a pipette it can be sealed again.   Sinton found that a temperature of 35° to 38° C. gave uniformly successful results.

As regards the cultivation of piroplasmata, there is little to add to what has been said above (p. 1028).   The technique is the same as that employed for malarial parasites.   Multiplication takes place by the usual budding method, and it appears that new red cells become infected, some of which may show as many as sixteen parasites.

Cultivation of Flagellates from Insects.

The various media which have been described above for the cultivation of flagellates from the blood and tissues of vertebrates can be employed for growing the closely allied insect flagellates.   In the case of insects, however, there is difficulty in obtaining the flagellates free from bacteria. The only forms which have been cultivated are those of the intestine, which has to be dissected out without bacterial contamination.   If the intestine contains bacteria which will grow in the medium, there is still greater difficulty in isolating the flagellates, though this can be done.

The dissection of insects is carried out with all precautions necessary to prevent bacterial contamination, sterile instruments, slides, and solutions being used.   Before dissection the insect can be immersed for a few seconds

in Weigert's iodine solution, placed in a sterile solution of hyposulphate of soda to remove the iodine, and then washed in sterile saline, from which it is transferred to the slide. By cutting open the body with sterile needles the intestine is drawn out. As recommended by Hoare for cultivation of *Trypanosoma melophagium* of the sheep ked, the intestine is then lifted up with sterile forceps and a large volume of sterile saline solution poured over it. The intestine is then transferred to N.N.N. or one of the other blood-media.

Nöller has employed another method, which depends upon the property rosettes or clusters of flagellates have of adhering to a slide. After the intestine of the insect has been dissected out as described above, it is opened and the flagellates liberated. The fluid containing them is transferred to a sterile cover-slip and left for some time. The clusters of flagellates sink and become adherent to the cover-glass, which is then washed with sterile saline which is allowed to flow over it. In this way bacteria are removed, and some, at least, of the clusters of flagellates remain adherent to the cover-glass. A drop of sterile fluid is then placed on the cover-glass, and the flagellates dislodged by a platinum loop. The fluid is then inoculated to the medium.

Some insects, especially fleas, as described above (p. 350), have the habit of voiding fæces during the feeding act. Such fæces can be received directly on to sterile portions of a cover-glass held behind the insect and transferred to the medium. The writer on several occasions has obtained bacteria-free cultures of leptomonas of fleas in this way.

In the case of certain insects like house flies, which are omnivorous feeders, the intestinal contents contain numerous bacteria. Advantage in these cases may be taken of the peritrophic membrane, within which the ingested material is confined (Fig. 175). Sometimes flagellates occur in the endotrophic space between the membrane and the intestinal wall. Bacteria appear to be absent from this space. If the intestine be removed without outside bacterial contamination, as described above, its wall can be opened at one point without damage to the peritrophic membrane, and cultures commenced from the fluid which escapes from the peritrophic space.

Cultivation of Coprozoic Protozoa.

The coprozoic Protozoa are those which develop in fæces as a result of cysts which have passed unharmed through the intestine. If fæces, especially those of herbivorous animals, be kept for a few days, Protozoa of various kinds appear in them. The fæces of pigs and frogs, for example, if kept moist, give rise to very rich cultures of coprozoic organisms. Amœbæ belonging to the genera *Hartmannella*, *Sappinia*, and *Dimastigamœba*, shelled amœbæ of the genus *Chlamydophrys* and its allies. flagellates belonging to the genera *Bodo*, *Spiromonas*, *Cercomonas*, *Heteromita*,

and *Copromonas*, and even ciliates, are the forms most usually met with. Many of these will grow and multiply on agar plates, where they feed upon the bacteria which develop. If freshly-passed fæces is streaked on such a plate, in a few days cultures will often be obtained. The medium which has been very extensively used is one which was devised by Musgrave and Clegg, and modified by Walker:

Agar	2·5 grams.
Sodium chloride	0·05 grams.
Liebig's beef extract	0·05 ,,
Normal sodium hydroxide solution	2·0 c.c.
Distilled water	100 c.c.

After solution has taken place, the mixture is distributed in test-tubes in quantities sufficient for making plates, as in bacteriological work. The material from which the culture is to be made is streaked on the plate in two or three parallel lines. The plates, which are kept with the agar upwards to prevent drops of condensation water falling on to it from the lid, are maintained at the laboratory temperature or in an incubator at about 22° C. Bacteria multiply rapidly, and commence to grow across the plate from the inoculated streaks. The Protozoa at first multiply on the original streak, but they gradually make their way over the plate in the direction of the spreading bacterial growth. By examining the plate with a low-power objective, the Protozoa can be detected in the bacterial growth as small refractile bodies. By scraping off some of the growth with a platinum loop and smearing on other plates, subcultures can be obtained. It is possible by the use of a fine capillary tube, heated to form a tiny bead at the end, to pick up individual amœbæ and transfer them to a fresh plate. In this way pure strains can be isolated.

Many coprozoic amœbæ and flagellates can be grown in weak solutions of egg albumen, in a liquid obtained by boiling fæces in water, or in hay infusion. In the liquid media the amœbæ are confined chiefly to the surface scum.

It has to be remembered that the mere occurrence of a recognizable stage of a parasite in fæces is not in itself an indication that it is a parasite of this particular animal. Oöcysts of species of *Monocystis* are common in fæces of birds which have fed on worms, while those of fish coccidia may appear in the fæces of human beings (see pp. 851 and 1147).

Maintenance of Parasitic Protozoa in Laboratory Animals.

This method of keeping strains of parasitic Protozoa in laboratory animals is in certain respects comparable with that of cultivation, for in many cases the organisms are maintained in animals which are not their natural hosts. Many strains of trypanosome—the pathogenic trypanosomes—will multiply and survive in rats, mice, guinea-pigs, and

other animals. This applies particularly to *T. gambiense*, *T. brucei*, *T. rhodesiense*, *T. congolense*, *T. evansi*, and *T. equiperdum*. If a rat is inoculated subcutaneously or, better, intraperitoneally with a drop of blood from an infected animal, trypanosomes appear in its blood after a varying incubation period. They multiply progressively, and finally bring about the death of the animal. Towards the end of the illness, and before or immediately after its death, some of its blood is inoculated to another animal, which acquires the infection. In this way strains can be maintained indefinitely. After several passages in one kind of animal the virulence of a strain is as a rule increased up to a maximum, the animals then surviving a fairly constant number of days, which for mice is about five, rats ten, and guinea-pigs twenty. By changing the kind of animal from time to time the virulence may be diminished. The inoculations may be carried out by abstracting a few drops of blood from the ear or tail of the animal, mixing it with a small quantity of physiological saline solution in a watch-glass, and injecting the mixture with a hypodermic syringe. A very simple method which can be used when only a small quantity of blood is to be injected is to use a piece of glass tube drawn out to a very fine capillary. The blood is allowed to run directly from the exuding drop into the capillary portion of the tube, which is immediately used for the inoculation as a needle, the blood being expelled by blowing with a rubber teat or the mouth. The manipulations must be carried out rapidly before coagulation has occurred. At the first inoculation into a laboratory animal the type of infection produced varies with the trypanosome used. Thus, rats inoculated from human beings infected with *T. gambiense* acquire a mild infection. Trypanosomes may be found only occasionally in the blood, the animals surviving for several months or even a year. In order to establish such a strain in the rat, it is advisable to sub-inoculate intraperitoneally with a large quantity of blood when trypanosomes are first seen to be present. By repeating the process, the virulence can be increased in five or six passages till the inoculated animals show a regular and progressive multiplication of trypanosomes in the blood, even when a single drop of blood or even less is inoculated. Other trypanosomes, like *T. brucei* or *T. rhodesiense* and *T. evansi*, are more virulent to rats at the first inoculation, though increase in virulence occurs after several passages. In the case of *T. cruzi* there is a different state of affairs. Rats, mice, and guinea-pigs can be infected, but less readily than with the pathogenic trypanosomes of Africa. Young animals are more susceptible. After several passages in one kind of animal the virulence diminishes, the animals tending to recover from the infection. Finally, inoculation may fail to infect. By using only young animals and sub-inoculating immediately trypanosomes appear in the blood, it may be

possible to maintain a strain indefinitely. This is facilitated by changing the kind of animal from time to time.

Trypanosomes of birds can in certain cases be kept by inoculation. Thiroux has shown that the naturally occurring trypanosome (*T. paddæ*) of the Java sparrow can be inoculated from canary to canary (p. 577). Blood can be obtained from a bird by puncture of a vein on the under-surface of the wing with a sharp needle. Inoculations are readily made into the pectoral muscles.

As regards *Leishmania donovani* and *L. tropica*, there is considerable difficulty in keeping strains in animals, owing to the decline in virulence in successive passages. To bring about infection in rats, mice, dogs, or monkeys it is necessary to inoculate intraperitoneally large doses of the parasite. The infection acquired may be a slight one or a heavy one. At each sub-inoculation a large dose is necessary, so that as a rule after at most a few passages the parasites are present in numbers too small to bring about infection. The recent work on the hamster appears to show that this animal is more susceptible, and that a strain can be maintained in it (see p. 416). In the case of *L. tropica* it is probable that a strain could be maintained indefinitely in the monkey or dog by inoculation into the skin. The animals recover from the infection, so that sub-inoculations must be made when the lesion appears to have reached its maximum size. The sore is excised or scraped, and the material containing parasites is injected into the skin of the next animal.

Strains of *Entamœba histolytica* can be kept in kittens. The primary infection can be secured by introducing into the stomach of an animal material rich in encysted forms or by injecting material containing the vegetative forms *per rectum*. The injection is best carried out by fitting to a syringe about 6 or 7 inches of a medium-sized catheter, which, with the aid of vaseline, can be passed into the stomach or large intestine into which the material is injected. The animals acquire an acute dysentery associated with the presence of a large number of amœbæ. The chances of such an infection occurring after rectal injections are considerably increased if the anus is sealed after the injection by a plug of cotton-wool soaked in a solution of collodion in ether. It is softened with ether and removed in two or three days, when a large infection can usually be detected. To sub-inoculate it is best to kill the animal, wash out the contents of the large intestine with warm saline solution, and even scrape the mucosa. The material so obtained is inoculated to the next animal. In this way it is possible to keep a strain indefinitely. According to Dobell (1926), the allied *E. nuttalli* of monkeys can be maintained in the same way.

The malarial parasite of birds (*Plasmodium præcox*) can be maintained indefinitely in canaries and other susceptible birds by inoculating blood

from one to the other. There is a marked difference in the susceptibility, some birds acquiring heavy and fatal infections, and others mild ones from which they recover.

Another parasite which is inoculable to a variety of laboratory animals and can be maintained in this way is *Toxoplasma gondii*, and the possibly identical forms found in other animals.

In many cases in which it is desired to maintain strains it is necessary to use the natural hosts, owing to the marked specificity of the parasite. The common trypanosome of the rat (*T. lewisi*) cannot infect any other animal under natural conditions. It can readily be inoculated from rat to rat, and a strain inoculated from a naturally infected wild rat into a white rat can be kept in this animal. An infection appears in the blood in six or seven days, reaches a maximum, and gradually subsides, so that sub-inoculations must be made during the period when trypanosomes are present.

The maintenance of strains in the natural hosts is of general application, and only necessitates a supply of animals which are known not to have been previously infected. This applies not only to parasites which can be infected by inoculation of blood, such as the malarial parasites of monkeys, the piroplasmata, and trypanosomes, but also to those in which infection results from the ingestion of encysted or other stages, such as coccidia, and even those which require to be injected by an intermediate host. In nearly all cases, in order to study the complete life-history of a parasite, experimental infection of the natural hosts is essential, and in order to carry out a sufficient number of observations, the successive infection of a number of these is necessary.

Not only are parasites maintained in vertebrates, but they can also be kept in invertebrates. In the case of those which are the intermediate hosts of vertebrate parasites, it is not usually convenient to keep the parasites in them, as they cannot, as a rule, be infected directly from one another by inoculation. Sometimes, however, when once the invertebrate is infected by allowing it to feed on the vertebrate, the parasite may survive in one individual for a long time. Thus, reduviid bugs, which may live for many months, and even bed bugs and ticks, may be infected with *T. cruzi*, which, without injuring the invertebrate in any way, survives in it for the rest of its life. The fæces of the bugs or the crushed-up intestine will produce infection in a suitable vertebrate at any time. In this case a trypanosome which is difficult to maintain in the vertebrate may be kept for long periods in the invertebrate, which happens to be very long-lived. Another illustration is afforded by *Babesia canis* of dogs. This piroplasm is readily inoculable from dog to dog, in which it often produces a rapidly fatal infection, so that to maintain·

a strain numbers of animals are required.   Ticks which will remain infective for long periods can be fed on a dog, and the parasites maintained in them.   On one occasion the writer collected ticks off a dog infected with *B. canis*, and produced infection in another dog six months later by allowing the ticks to feed on the animal.   Other species of *Babesia* behave in a similar manner.   By breeding *Lynchia maura* in cages in which pigeons were kept, the writer has maintained a strain of *Hæmoproteus columbæ*. A fresh pigeon introduced from time to time becomes infected by the flies which feed on it.

Parasites which are peculiar to invertebrates can often be maintained in their hosts.   This is usually done by allowing them to breed, care being taken to introduce to the breeding-cage a certain number of individuals already infected.   Rat fleas infected with flagellates can be reared in this way in a cage with a rat.   The fleas become infected from one another, so that a constant supply of infected insects is at hand.   The method is applicable to any invertebrates which can be reared in captivity.

Importance of obtaining Clean Hosts for Experimental Infections.

It has already been mentioned that in order to study the complete life-history of a parasite it is advisable, whenever possible, to study the infection in an animal which has been infected experimentally.   In this way definite information regarding the rate of development, the duration of the infection, and the successive stages of the life-cycle can be obtained. It is essential that clean or uninfected animals be used.   In the case of vertebrates there is often considerable difficulty in obtaining such animals, reliance having to be placed on repeated negative examinations of the blood and fæces.   It is possible, by breeding from animals in which no signs of infection can be discovered, to obtain batches which are free from infection.   The criterion of freedom from infection varies with different parasites.   The mere failure to discover parasites by direct examination may not be sufficient, as shown by the frequency with which parasites can be cultivated from the blood when none is actually visible, and by the possibility of producing infections in other animals by inoculation of blood.   In the case of trypanosomes of cattle and sheep and leishmania in man culture from the blood will often reveal infections not otherwise detectable.   It is well known that after an acute attack of piroplasmosis the blood of an animal remains infective to inoculated animals, sometimes for years, though no parasites can be found in the blood.   These illustrations serve to indicate how difficult it is to exclude absolutely a previous infection unless it has been possible to observe the animals from their birth, or to obtain them from localities in which the particular infection

does not occur.   In certain cases it has been claimed that infections can be eradicated and animals rendered clean by drug treatment.   Thus, during his experiments on the infection of rats and mice with the human intestinal amœbæ, Kessel concluded that the naturally occurring amœbæ of these animals could be got rid of by dosing the animals with magnesium sulphate on two successive days (see p. 206).

Even if it can be concluded with tolerable certainty that an animal is free from infection, unless it has been observed from birth it may have had the infection previously, and in some cases this will have conferred upon it an immunity to reinfection.   Rats which have once been infected with *Trypanosoma lewisi* cannot be reinfected, and this appears to be true of many of the coccidial infections.   Failure to infect an animal with a parasite may be due to the fact that previous infection has occurred.   On the other hand, some hosts appear to acquire no immunity, and can be repeatedly infected, as is the case with the trypanosome of sheep, and probably the intestinal flagellates and amœbæ.   From the above remarks it follows that for the study of the life-histories of parasites, the safest method is to use young animals which have been carefully observed, and for preference those whose parents were apparently free from infection.

The general principles outlined above apply very largely to invertebrates also.   In the case of these animals, it is usually possible to breed clean offspring.   The eggs, larvæ, or pupæ can be isolated, and the resulting individuals may be exposed to infection as desired.   Such uninfected invertebrates are very necessary for the study of the development of blood-parasites of vertebrates.   For example, tsetse flies in nature are liable to infection with several different trypanosomes.   It was only when observers adopted the method of hatching flies from pupæ and allowing them to feed on vertebrates infected with a known trypanosome that the cycles of development of the various forms in the fly could be satisfactorily elucidated.   By employing flies bred in the laboratory, Kleine first demonstrated the fact that they do not become infective till after the lapse of about three weeks from the time they feed on infected animals.   The developmental forms of the various malarial parasites of man and birds in mosquitoes are very similar to one another.   By employing mosquitoes hatched from pupæ it was discovered that the human parasites would develop only in species of *Anopheles* and the bird parasites only in species of *Culex* and their allies.   Another illustration is afforded by the sheep trypanosome.   The ked which lives on sheep always has an intestinal flagellate infection.   It was supposed that this was a flagellate peculiar to the ked.   Kleine hatched keds from pupæ, and allowed them to feed on goats.   No intestinal infection was acquired. Only those which fed on sheep developed the infection, a fact which helped

to prove that the flagellate infection was acquired from the trypanosome of sheep.

In the case of the various species of *Babesia*, there is considerable difficulty in obtaining clean ticks for experimental purposes, for these parasites pass through the egg of the tick and render the succeeding generation infective. In such cases it would be necessary to prove the non-infectivity of adult ticks by allowing them to feed on susceptible hosts. If these did not become infected, it might be safe to assume that the ticks were not infected. They could then be used for raising clean offspring. In any case there is considerable uncertainty, and it is probably on this account that knowledge regarding the development of species of *Babesia* in ticks is so imperfect.

Insects and other invertebrates are often infected with parasites peculiar to themselves, and, as in the case of parasites which are limited to vertebrates, it is often necessary to obtain uninfected hosts which can be experimentally infected. This, again, is most readily accomplished by separating the eggs and rearing batches which have not been exposed to infection.

Observations on Killed Protozoa.

It not infrequently happens, especially in the case of flagellates and ciliates, that their movements are so energetic that certain details cannot be observed. Without any attempt at permanently fixing or staining them, much information can be gained by killing them by exposing the fluid on the slide to osmic acid vapour for ten to fifteen seconds. The drop of fluid on the slide is simply inverted over a bottle containing a 1 per cent. solution of osmic acid. Another method is to mix the fluid with a drop of iodine solution, which has the advantage of colouring the organisms a faint brown. Both these procedures not only kill the organisms, but bring about fixation, so that structures such as flagella or cilia and nuclei are more clearly seen (see p. 308). The iodine method is of great value for the examination of encysted Protozoa, especially in fæces, for the nuclei, which are with difficulty detected in living cysts, and vacuoles containing glycogen which stains a brown colour, become clearly visible (Plate II., p. 250).

PERMANENT PREPARATIONS OF FIXED AND STAINED PROTOZOA.— Permanent preparations of fixed and stained Protozoa are essential for the study of many of the finer details of structure. Practically all the methods used in cytological work have been applied to the Protozoa, and many special methods have been introduced, but it is only necessary to consider a few of those which are of more general application.

Preparation of Dried Films.—This method of examination is considered first, not because it is one which gives accurate information regarding

the structure of organisms, but because it is simple of application and widely used for diagnostic purposes. As regards the structure of the cytoplasm and the nucleus it is most misleading. Nevertheless, it will frequently give an indication of the general shape of an organism, the position of the nucleus and other structures in the body, the number of flagella present, and other details. When the appearance of known organisms prepared in this way has been observed, it is possible to recognize the actual species. The method is thus very useful for purposes of identification of certain trypanosomes, malarial parasites, leishmania, and even intestinal parasites, and when used in conjunction with other more accurate methods may yield valuable information.

The method consists in making thin films on slides which must be free from grease. The film is dried as rapidly as possible by waving it in the air. A very common method of making the film is to take a small drop of fluid on the end of one slide which is pushed along the surface of another slide at an angle of about 45°. Blood-films are made in this way, as also films of any liquid. Films of solid organs such as the liver or spleen are made by simply smearing a piece of the organs lightly across the slide or merely dabbing the slide with the freshly-cut surface. Thin films of intestinal contents can be made in a similar manner. It must never be forgotten that the operation of film-making breaks up many of the larger cells which are present. Portions of these (fragmentation bodies) are present in the films, and have frequently given rise to confusion when their origin has not been recognized. The process of smearing and drying undoubtedly distorts and may disintegrate the parasites themselves. This may be avoided to some extent by exposing the film to osmic acid vapour for ten to fifteen seconds before allowing it to dry. If an attempt be made to dry a film of liquid containing salt, as the water evaporates the concentration of the salt increases, and this has a disastrous effect on the organisms. In such cases the drop of liquid may be placed on a slide and exposed to osmic vapour before the film is spread. The osmic killed and fixed organisms are much less liable to distortion.

When the film is dry, if a watery stain is to be used, it is fixed by immersion in a cylindrical glass bottle containing absolute alcohol, in which it is left for about five minutes. It is then taken out and allowed to dry as it leans against a support in an almost vertical position. The film can then be stained. This is most satisfactorily carried out by one of the modifications of the Romanowsky stain, which has the property of colouring cytoplasm blue and chromatin granules, flagella, red blood-corpuscles, and other structures various shades of red or purple. Simple stains, such as methylene blue or hæmalum, can be employed, but these do not give such good differential staining or contrast of colours.

Giemsa's Stain.—To stain a film with Giemsa's Romanowsky stain, it is placed in a dish film-side downwards, and supported at each end on pieces of fine glass tubing or on ridges which are present in the dish. To 10 c.c. of acid-free distilled water ten drops of the stain are added in a graduated tube. Mixture is effected by inverting the tube, and the diluted stain is poured into the dish so that it passes under the slide. The dish is covered, and staining is continued for ten minutes or longer according to the intensity required. For fine objects such as flagella it may be left for one or more hours. The film is then removed and washed in running distilled water for about a minute. It is then dried, either by leaning it in an inclined position or blotting it with smooth filter-paper. The film is then ready for examination. If an oil-immersion lens is not used, it will be necessary to cover the film with a thin layer of immersion oil. For examination with the oil-immersion lens oil is placed directly on the film. After examination the oil is removed by running xylol over it and allowing it to dry, in which condition it can be kept. The film can be mounted in Canada balsam with a cover-glass, but unless the balsam is definitely neutral fading rapidly takes place.

Leishman's Stain.—Another method of staining films is by Leishman's modification of Romanowsky stain. This stain, being a solution of the colouring matter in methyl alcohol, can be used for fixation as well as staining. The dried and unfixed film is placed on a support in a horizontal position with the film-side upwards, and five to ten drops of the alcoholic stain are dropped on to it. It is left for a half to one minute, and then distilled water is added in a volume which is double that of the stain used. Mixture is effected by gently rocking the slide or by moving a glass rod in contact with the surface of the liquid. The staining occupies from five to ten minutes or longer. When this is completed, the slide is flushed with distilled water, care being taken not to pour the stain off in such a way that the surface scum which has formed settles on the slide. Distilled water is left on the slide for a minute, after which it is poured off and the slide dried.

Defective Staining.—It not infrequently happens, especially in the tropics, that the above stains fail to give satisfactory results. In most cases, provided the stains are not too old and have been kept in properly stoppered bottles, so that they have not absorbed moisture, this is due to defects in the alcohol or the distilled water. The alcohol employed for fixing the films for Giemsa staining must be absolute alcohol free from acid, and the same remark applies to the methyl alcohol used for dissolving the Leishman stain. The commonest source of error is, however, the distilled water, which must be quite neutral and free from acid. A simple method of testing water is to add to a quantity in a test-tube a

very small granule of bromo-cresol purple. If the water is acid it will form a pale yellow solution, while if alkaline it will be purple. The reaction of the water should be such that it gives a purple colour, which is turned to yellow by the slightest trace of acid. If the water is acid and gives a yellow colour, a solution of carbonate of soda (1 in 1,000) can be added drop by drop to about 100 c.c. of the distilled water till the purple colour appears. Excess of soda solution must not be added. The coloured water can be used for the staining process without ill-effects.

The best results are obtained with newly-prepared films. Blood-films and tissue smears, if not stained within a day or two of making, begin to deteriorate, especially in the tropics. With increasing age the staining becomes more and more defective, and though methods have been devised for rejuvenating old films, none of them gives the results which can be obtained by staining them soon after they are made. The defective staining of old films is seen in the uniform blue coloration of the red blood-corpuscles. This can be remedied to some extent by extracting the blue by treating the stained film with a 1 per cent. solution of acid sodium phosphate.

Fallacies Due to Contamination.—It will be advisable to consider here the fallacies which may arise in connection with the making and staining of dried films. Many of these have been referred to above. They are the result of extraneous organisms and other objects finding their way into the films, and being regarded as belonging to the blood or other material examined.

1. The slides may be dirty, and have upon them bacteria, yeasts, or other organisms. If previously used for film work, the remains of a previous film may be present if they have been improperly cleaned. Scratches or fissures on the surface may give rise to appearances difficult to interpret owing to the deposit of stain. Slides which are allowed to lie about quickly become contaminated from the air, while flies may deposit fæces containing flagellates.

2. The alcohol used for fixing may have become dusty, so that foreign particles adhere to the slide, and the same will apply to the stains.

3. Contaminations readily occur when the film is actually made. If blood is taken from an imperfectly cleaned finger or other area of skin, bacteria and yeasts may be present. The tail of an animal, such as a rat or mouse, is a very fruitful source of such cutaneous contaminations. Similarly, when films are made *post-mortem* from the blood and organs, outside contaminations are common. If the intestine has been opened before films are made, contamination with bacteria from the intestine commonly occurs. Films made in the field from animals which have been shot are almost invariably contaminated either from the skin or wounded intestine with bacteria, spirochætes, or Protozoa.

4. When films are made some time after the death of an animal, various bacteria, yeasts, and even flagellates may be present, owing to the fact that they have entered the blood and tissues from the intestine either shortly before or after death.

5. If a film has been made and is left exposed, dust, including bacteria and yeasts, readily falls on it from the air. Fungus spores will germinate and grow across the slide as filaments. House flies will feed on the film, and often produce a worm-eaten appearance. They may also contaminate the film with bacteria or with their fæces, which not infrequently contain flagellates and other organisms. The deposits of fæces may be spread across the film by the legs or proboscis. When the film is stained, the various objects deposited may appear as if they were present in the original material.

6. The distilled water used is a very common source of error. It very soon becomes contaminated with bacteria and Protozoa, especially in the tropics. When such water is used for diluting the stain, the organisms are killed and stained, and, especially when Leishman stain is employed with the film-side upwards, they sink on to the film, to which they adhere as variously stained bodies.

7. After a film has been stained, though bacteria and yeasts may fall on it, they are unlikely to cause confusion, as they are unstained. On the other hand, stained objects may be transferred from one slide to another by blotting-paper used for drying films in succession.

Preparation of Wet Fixed Films.—Films which have been made on slides or cover-glass may be fixed before they are allowed to dry with one of the fixing fluids which experience has shown to be good for cytological work. After fixation the films are washed free of fixative, and stained and mounted like sections fixed on slides. No drying is allowed to take place at any stage. The results obtained by wet fixation are far superior to those given by any of the drying methods described above. It is applicable to blood and tissue films, films of intestinal contents or fæces, the centrifuged deposits from urine or other liquids, the tissues or intestinal contents of insects, the fluid from cultures, the growth on agar plates, and, in fact, any material in which Protozoa occur. It is very largely used for the study of intestinal Protozoa of man and animals, and is the best method for making permanent preparations of these organisms. It has to be remembered that many of the fallacies, such as the occurrence of extraneous organisms and the disarrangement of the tissues, occur as when dried films are made.

The procedure is to make a thin film of the material on a slide or cover-glass, preferably the latter, and drop it immediately, film-side downwards, on to the surface of the fixing fluid in a Petri or other suitable dish. If

the material is thick, it should be first emulsified with physiological saline solution. In the case of blood and tissues, fæces, even when emulsified with saline, fluid from blood or serum culture media, or any liquid containing albuminous matter, the film will as a rule adhere to the cover-glass, and will remain there throughout the subsequent manipulations if care be taken. If films are made from the centrifuged deposit of urine or water, they will not adhere unless some adhesive is used. Adhesion can be effected by placing on a cover-glass side by side a drop of the material and a drop of serum. The two are rapidly mixed and spread over the cover-glass, which is at once dropped on to the fixing fluid. A cover-glass usually floats on the surface of fixative. If it sinks, it should be turned over after fixation to prevent the film being rubbed off. As fixative, one of the following can be used:

Schaudinn's Fixative.—This is prepared by adding one volume of absolute alcohol (or 96 per cent. alcohol) to two volumes of saturated aqueous solution of mercuric chloride. This mixture is kept as a stock solution. Immediately before use, it is advisable to add acetic acid to the quantity to be used to the strength of 5 per cent.

Zenker's Fixative.—The following solution is made:

Mercuric chloride	..	..	..	..	.. 5 grams.
Potassium bichromate	..	..	..	..	.. 2·5 ,,
Sodium sulphate	..	..	..	..	.. 1 ,,
Distilled water	..	..	..	..	.. 100 c.c.

This is a stock solution, and, as in the case of Schaudinn's fixative, acetic acid to a strength of 5 per cent. is added to the quantity to be used.

Bouin's Fixative.—This consists of the following mixture:

Picric acid saturated aqueous solution	..	..	30 parts.
Formol (40 per cent.)	..	..	..	.. 10 ,,
Acetic acid	..	..	..	..	.. 2 ,,

The various solutions are best mixed when the fixative is required for use.

Alcoholic Bouin's Fixative.—This has the following composition:

Alcohol (80 per cent.)	..	..	..	..	.. 150 c.c.
Formol (40 per cent.)	..	..	..	..	.. 60 c.c.
Acetic acid	..	..	..	..	.. 15 c.c.
Picric acid	..	..	..	..	.. 1 gram.

A stock solution of the picric acid in the alcohol can be kept, and the other ingredients added in the correct proportions to the quantity required for use at any moment.

Of these four fixatives, the Bouin's mixtures are probably the simplest to use, as the picric acid is more easily washed out after fixation than the mercuric chloride in the others. Fixation of films is complete in fifteen to thirty minutes. It is more rapid if the solutions are warmed.

If Schaudinn's fixative has been used, after fixation is complete the films are transferred to 70 per cent. alcohol, in several changes of which they are washed in order to remove the sublimate. Finally, they are washed in alcohol of the same strength, to which a few drops of Weigert's iodine solution has been added, the mercuric chloride being converted into the more soluble mercuric iodide. The films which have been stained yellow by the iodine are again transferred to 70 per cent. alcohol to which a drop of 1 per cent. solution of sodium thiosulphate ("hypo.") has been added in order to remove all traces of iodine. During these processes a stay of a few minutes in each solution will be sufficient. The films may then be transferred to 96 per cent. alcohol, in which they can be left for some hours to harden. This is, however, not a necessary procedure, though it may be employed if the films have to be left for a day or two before staining. As the majority of stains are watery solutions, it is necessary to bring the films into distilled water, and this is accomplished by passing them through changes of alcohol of decreasing strength (70 per cent. alcohol plus one part of water, 70 per cent. alcohol plus two parts of water, and 70 per cent. alcohol plus three parts of water) and then into distilled water.

After fixation with Zenker's fluid, which does not contain alcohol, washing is carried out with distilled water, the iodine and sodium thiosulphate solutions being added to it.

After Bouin's fixative, the films are merely washed in changes of distilled water till all the picric acid has been extracted. If alcoholic Bouin is used, washing is commenced in 70 per cent. alcohol and the films are taken down to distilled water through graded alcohols.

The films, when in distilled water, can be stained by any of the methods employed for staining sections. The following will give satisfactory results:

Mayer's Hæmalum :

Hæmatein	1 gram.
Alcohol (90 per cent.)	50 c.c.
Alum	50 grams.
Water	1,000 c.c.

The solution is prepared by dissolving the hæmatein in the alcohol with the aid of heat, and adding to the solution the water in which the alum has been dissolved. A crystal of thymol may be placed in the bottle to prevent growth of fungi or other organisms.

To stain films they are placed in the stain, which is best diluted at least twenty times. The best result is probably obtained by adding a few drops of the stain to a Petri dish of distilled water, and leaving the films in for several hours or overnight. After staining, the films are washed in running tap water till they are quite blue. They are then taken through alcohols

of increasing strength up to absolute alcohol, in two or three changes of which they are dehydrated.   Finally, they are cleared in xylol and mounted in balsam.

Mayer's Acid Hœmalum.—This stain is of the same composition as Mayer's hæmalum, except that it contains acetic acid.   It can be prepared from it by adding acetic acid to a strength of 2 per cent.   It is used in the same manner, but has less tendency to over-stain.   On this account it is more precise.

If over-staining appears to have occurred with either of these stains, it can be remedied by placing the films in acid alcohol (70 per cent. alcohol to which hydrochloric acid is added to a strength of 1 per cent.).   After being decolorized, the films must be well washed in running tap water till the acid is completely neutralized and they are blue.

Heidenhain's Iron Hœmatoxylin.—This stain involves the use of two solutions—the actual staining fluid and a mordant.   The stain is prepared by dissolving 1 gram of hæmatoxylin in 10 c.c. of 90 per cent. alcohol with the aid of heat, and adding to the solution 90 c.c. of distilled water. This is allowed to ripen for a week or ten days, after which an additional 100 c.c. of distilled water are added.   The mordant consists of a 4 per cent. solution of the violet crystals of iron alum (sulphate of iron and ammonium) in distilled water.

The films are placed in the mordant for several hours, quickly rinsed in distilled water, and placed in the stain, where they become black.   They are left in the stain for several hours, and, after washing in distilled water, are transferred to the mordant diluted to a 1 per cent. solution with three parts of distilled water.   The black deposit is gradually dissolved by the excess of iron alum, first from the cytoplasm of the cells and then from the nuclei.   This decoloration or differentiation is continued till the granules in the nuclei are distinct, the films being removed into distilled water and examined on a slide with the microscope from time to time.   When differentiation is complete, the films are washed in several changes of distilled water and then for half an hour or more in running tap water. They can be then counter-stained with a 1 per cent. solution of eosin or orange G.   The films are then dehydrated by passing them through graded alcohols, cleared, and mounted as described above.   With this stain the correct degree of differentiation varies according to the particular object in view, and must be determined by direct observation.   Small objects such as trypanosomes require only a few minutes, but larger ones, like amœbæ or their cysts, ten minutes or more.   If too much stain has been extracted, the films can be re-stained by placing them again in the mordant and repeating the various processes.

As a modification of the above method, alcoholic solutions may be used.

The mordant is prepared just before use by diluting 1 part of the 4 per cent. aqueous solution of iron alum with 10 parts of 70 per cent. alcohol. The stain is similarly prepared by diluting 1 part of the stain with 10 parts of 70 per cent. alcohol. If films have been fixed in an alcoholic fixative, such as Schaudinn's fluid or alcoholic Bouin's fluid, all the washing is carried out in 70 per cent. alcohol. The film is then placed in the mordant, which is kept warm in the incubator for fifteen to thirty minutes. It is then rinsed in 70 per cent. alcohol and placed in the warm stain for about thirty minutes to an hour. Differentiation is made in the alcoholic mordant, after which the films are washed in several changes of 70 per cent. alcohol, dehydrated, cleared, and mounted. The advantage of the method is that the necessity of bringing the films into water after fixation is eliminated. Though it is more rapid than the longer method, and frequently gives very excellent results, these are less regularly obtained. Furthermore, care must be taken to use only freshly diluted mordant, as the alcohol after some time causes a precipitate to form.

Romanowsky Stain.—The staining of wet fixed films by Romanowsky stain is much more difficult to accomplish than in the case of dry films. When it is satisfactorily carried out, the nuclei should be of a red or purple colour and the cytoplasm blue, as in dried films.

The films, which may be fixed by any of the methods described above, must be very carefully washed to remove all trace of mercury and acid. It is well to wash them for some time in running tap water before finally bringing them into distilled water for staining. The best results are obtained with Giemsa stain, which is used in a strength of 0·5 c.c. in 20 c.c. or even 50 c.c. of distilled water. The stain is allowed to act for several hours, or as long as twenty-four hours. During this time it is better to change the stain once or twice. When staining is complete the films must be dehydrated, but this cannot be done with alcohol as it extracts the stain. The films, after being rinsed with distilled water, are passed through the following mixtures of acetone and xylol before being cleared in pure xylol and mounted in balsam:

1. Acetone 95 c.c. : xylol   5 c.c.
2. Acetone 70 c.c. : xylol 30 c.c.
3. Acetone 30 c.c. : xylol 70 c.c.
4. Xylol.

Fixation in Liquids.—It not infrequently happens that Protozoa occur in liquids in numbers which are too small for the preparation of films directly. The organisms can in these cases be concentrated by the centrifuge, and films made with the addition of serum from the deposit. There is danger, however, that the organisms may be damaged by the

centrifuging operation. It is possible in these cases to add a large quantity of fixative to the liquid. The liquid can be placed in a test-tube to one-third its height, and filled with the fixing fluid. If it be allowed to stand vertically, the Protozoa will sink to the bottom. The supernatant fluid can be removed and the tube filled with water or 70 per cent. alcohol according to the fixative. Sedimentation is again allowed to take place, and by repeating the process many times with the various fluids and stains used for the preparation of wet fixed films the organisms can eventually be stained and brought into xylol. A small quantity of the final deposit is placed on a slide with balsam and mounted.

It is sometimes convenient to preserve, in bulk, material such as fæces containing Protozoa, especially the encysted forms. A quantity is emulsi-fied in saline solution placed in a test-tube and the fixative added. By sedimenting and washing many times all the fixative can be removed. The material is then brought into 70 per cent. alcohol, in which it can be preserved indefinitely. By use of the centrifuge the washing processes can be carried out more rapidly.

Preparation of Sections.— One of the most important methods of investi-gating the parasitic Protozoa is the cutting of serial sections of the tissues of the host or the hosts themselves. These are fixed and embedded in paraffin, and serial sections are mounted and stained by any of the stains described above for wet fixed films. No other method will give such an accurate picture of the true relation of the parasite to the cells of the host. As an example, the difference between the distribution of *Leishmania donovani* in smears of the spleen and in sections of the same organ may be mentioned. In the smear many of the parasites are extracellular, while others appear as if they are included in cells in which they do not actually occur. In sections practically all the parasites are intracellular, and occur only in the large macrophages. Similarly, by smearing the tissues of insects on slides, purely intestinal parasites may appear to occur in other situations, and even within cells, owing to parasites being superimposed on the drying cells. When sections have been made, parasites will not be found in any situations unless they occur there normally. On this account it is not safe to make deductions from smears unless control sections are also made.

The importance of making serial sections depends on the fact that certain stages of development of Protozoa are so large that an individual appears in several sections, and it has to be studied in all of these. The exact distribution of a parasite in its host is also best followed in serial sections.

When the hosts are large, it is only possible to fix small portions of any particular organ. With smaller hosts the whole organ may be dealt with,

while many small invertebrate hosts can be fixed and sectioned without any dissection.

The methods of sectioning the tissues of vertebrates are well known, and need not be described here. It is essential that fixation be carefully carried out if the true structure of the organisms is to be preserved. On this account tissues preserved in alcohol or formalin alone do not give satisfactory pictures of the Protozoa they may contain. Zenker's and Bouin's fluids give very good results. After fixation for about twenty-four hours the tissues are washed in running tap water for a similar period in the case of Zenker's fluid, and in several changes of 90 per cent. alcohol in the case of Bouin's fluid.

In the case of invertebrates, especially when they are small, there may be difficulty in obtaining the required organs. A knowledge of the anatomy of the host, the methods of dissection, and the appearance of the organs is essential to success. The invertebrates may be fixed entire, but, especially in the case of arthropoda with tough impermeable cuticles, there is the obvious disadvantage that fixation of the tissues is never so good as when the animal has been opened to allow penetration of the fixative or when the individual organs have been removed and fixed separately. For a host as large and tough as the sheep ked, Hoare obtained very satisfactory results for the study of the sheep trypanosome by fixing the insects in the following manner, which was recommended to him by Dobell:

The ked is dropped into the following modification of Bouin's fixative:

Saturated solution of picric acid in 90 per cent. alcohol ..	75 parts.
Formol (40 per cent. formaldehyde)	25 ,,
Acetic acid	5 ,,

To the quantity to be used one to two drops of chloroform are added. This tends to soften the chitin and helps penetration of the fixative, which is allowed to act for twenty-four hours, during the first of which it is warmed on the paraffin oven. The insect is then placed in 90 per cent. alcohol and left there for seven days, the alcohol being changed several times. It is then passed through the following solutions, in each of which it remains twenty-four hours:

1. Absolute alcohol.
2. Mixture of equal parts of absolute alcohol and chloroform.
3. Warm chloroform saturated with paraffin.

After this it is placed in pure paraffin in the oven at 56° C. for five or six hours and finally embedded. Serial sections are then cut, mounted, and stained.

The method is one which can be used for any arthropods with chitinous integuments, and it will be found that the Protozoa have been fixed very

satisfactorily. This method, in which the host has been fixed without any disturbance of its organs, affords a very useful control of the sections obtained by fixing the organs separately.

In certain instances it may be possible to remove part of the chitinous covering to allow of better penetration of the fixative. In the case of ticks, by careful dissection with fine scissors and needles it is possible to remove either the dorsal or ventral surface of the abdomen. The tick can then be fixed in Bouin's or Schaudinn's fluid as an ordinary piece of tissue. When fixation is complete, the whole of the contents of the abdomen can be removed as one mass, which is then embedded and cut.

The method can be used for obtaining sections of the salivary glands of mosquitoes. These organs lie in the ventral part of the thorax. With care, all the thoracic contents can be dissected out as one mass, which is fixed. In the serial sections it is not difficult to find the salivary glands (Plates VII. to X., p. 916).

Not infrequently it is advisable to remove the individual organs by dissection, and to fix and deal with them separately. The stomach of a mosquito infected with malarial parasites can be removed from the body and fixed in Bouin's or Schaudinn's fluid. It is then passed through the various fluids, and finally embedded in paraffin. Similarly, the intestine of any insect can be removed and fixed for the study of flagellate or other infections.

Dissection of Mosquitoes and Tsetse Flies for Detection of Infections.

In the case of certain hosts, such as mosquitoes and tsetse flies, which are known transmitters of disease, special methods of dissection for the determination of the presence or absence of a Protozoal infection have been elaborated.

Mosquitoes.—These insects are often dissected with the object of discovering malarial parasites on the stomach wall or in the salivary glands.

To obtain the salivary glands of a mosquito, the legs and wings are removed and the body is placed on a slide in a drop of saline. With a needle in each hand the operator holds the thorax with one needle, while the end of the other is placed between the head and thorax. By careful and steady traction the head is drawn away from the thorax, and in many cases, as the head separates, the salivary glands, which consist of three lobes on each side, are drawn out of the ventral part of the thorax still attached to the salivary duct. With the needle used for drawing off the head the salivary duct is cut and the glands liberated. If the glands do not come away with the head, gentle pressure on the thorax may express them, or they can be dissected out from the ventral part of the thorax

with the needles.   These and the subsequent operations can be performed
without a microscope, but they are very much simplified by the use of a
simple lens or a binocular dissecting microscope.   It is essential that the
operator be able to recognize the glands as soon as they appear.

The head and body of the mosquito are removed from the slide, together
with any other débris.   A cover-glass is then applied, and it is possible to
examine the flattened salivary glands with the microscope for sporozoites,
which can readily be detected with the one-sixth objective.

If it be desired to obtain stained preparations of sporozoites, it is better
to examine the glands without a cover-glass.   Even if a cover-glass has
been applied, it can be removed after the addition of a sufficient quantity
of saline solution.   If sporozoites are present, the fluid on the slide should
be removed by drawing it off with pieces of blotting-paper.   The salivary
glands should be left on the slide in the smallest possible quantity of fluid,
the rest of the slide being carefully dried.   With the point of a needle the
glands are then broken up to liberate the sporozoites.   The fluid is then
spread over an area about $\frac{1}{4}$ inch in diameter with the needle.   The slide
is then allowed to dry, or before drying it is dropped film-side down into
a fixative as described above.   The dried films stained by Romanowsky
stain give very beautiful pictures of the sporozoites (Plate XI., p. 916).
For the details of their structure the wet fixation is necessary.

To obtain the stomach or mid-gut, the body of the mosquito from which
the head has been removed can be employed.   It is placed in a drop of
saline, and with the point of a needle the edge of the chitin on the dorsal
and ventral side of the abdomen near its posterior end is cut, care being
taken not to incise it so deeply that the intestine is severed.   The thorax
is held with one needle while the point of the other is placed on the posterior
end of the abdomen behind the incisions.   Traction is made away from
the thorax.   The posterior portion of the abdomen separates, and the
unsevered intestine draws with it the stomach, which can be examined
for oöcysts with or without a cover-glass.

To obtain permanent preparations of the entire stomach, the following
method has given good results in the writer's hands: A cover-glass is
applied to flatten the stomach.   The quantity of fluid must be sufficient
to prevent rupture of the organ.   By drawing off the fluid after the cover-
glass has been placed on the slide, the required degree of flattening can
be obtained.   A very small drop of Schaudinn's or Bouin's fluid is then
placed at the edge of the cover-glass, and a corresponding quantity of saline
drawn from the opposite side.   The process is repeated till, by the change
in the appearance of the stomach, it can be seen that the fixative has
gained access to all parts of the organ.   It is necessary to avoid the addition
of a large drop of fixative at any one time, as this will raise the cover-glass

and permit the stomach to contract. When fixation is complete, a larger quantity of fixative is allowed to run in, so that the cover-glass is raised. In many cases the flattened stomach will be separated from the slide and remain attached to the cover-glass as it floats on the surface of the fixative, which is allowed to act for another ten or fifteen minutes. The cover-glass is then carefully raised and placed in 70 per cent. alcohol, and dealt with as a wet fixed film. For staining very dilute acid hæmalum gives good results.

Tsetse Flies.—These flies, as the transmitters of pathogenic trypanosomes in Africa, are often dissected. The various structures which make up the proboscis, the salivary glands, and the intestine are the parts which require examination.

Two methods of removal of the salivary glands have been used. In the one the glands are pressed out of the posterior cut end of the abdomen after the intestine has been withdrawn, in the other the glands are drawn out of the anterior end of the abdomen before removal of the intestine, which can be extracted later.

Posterior Method.—This method was employed by Bruce and his co-workers, and is used by Duke, to whom the writer is indebted for the details of the technique to be followed:

1. Cut off the legs and wings.
2. Cut off the last two or three abdominal segments.
3. Place the cut end of the abdomen in contact with the left edge of a drop of saline solution on a slide.
4. Holding the fly with the fingers of the left hand, anchor a portion of the protruding viscera with a needle held in the right hand.
5. By gently jerking the fly and the viscera by movements of the left hand and needle, the abdominal contents are withdrawn.
6. With a hand lens examine the viscera to see if the salivary glands have been removed with the intestine. As a rule they will have remained in the abdomen, a varying length protruding from the cut end.
7. If they have remained in the abdomen, they can be pressed out by holding the fly with the left hand, placing the needle across the junction of the thorax and abdomen, and moving the needle while maintaining pressure towards the cut end of the abdomen.

The glands can then be dissected out from other organs and tissues which are removed. A cover-glass is applied, and the gland examined with a one-sixth objective. In determining a salivary gland infection, great care must be taken to exclude free flagellates lying above or below the glands. Long trypanosomes are to be looked upon with suspicion in this respect.

For discovery of trypanosomes in the intestine, it may be cut into portions and the contents expressed.

Anterior Method.—This method for removal of the salivary glands is one which was elaborated by Lloyd, and has the advantage over the posterior method in that the salivary glands are obtained free from any adherent fat body or other tissues, while the danger of contamination from the intestine is reduced to a minimum, as the only lesion occurs in the anterior portion of the œsophagus. Lloyd (1912) and Lloyd and Johnson (1924) describe the technique as follows:

The fly is held firmly in the fingers, and a longitudinal incision is made in the median dorsal line of the thorax from the neck to the abdomen. The insect is then immersed in normal saline salution, and incisions are made along the transverse groove of the thorax from the median incision almost to the bases of the legs. The strong muscles in the thorax, which run in a longitudinal direction, are also severed. A needle is now placed in the anterior end of the longitudinal incision and another in the posterior end. A gentle longitudinal pull applied to the fly by these needles causes the remainder of the thorax to break across. The alimentary canal breaks between the pharynx and proventriculus, while the salivary glands are drawn out of the abdomen quite free from fat body, and with only the finer twigs of the tracheal system adhering. The remainder of the thorax and head are now dissected away to leave a preparation consisting of proboscis, pharynx, and salivary glands. The intestine can be drawn out from the abdomen by the posterior method after removal of the salivary glands.

For examination of the proboscis for trypanosomes, its various parts have to be separated. First the labium is drawn away from the labrum and its bulb by placing the points of the needles on the base of the labium and bulb respectively. The hypopharynx may remain in the groove of the labium, or it may still be within the labrum. In the latter case it must be removed from the labrum, and this is again done by pressing on the bulb with one needle while with the other the hypopharynx is drawn out. The whole of the labrum with its bulb is then cut off. The dissection of the proboscis can be carried out after the salivary glands have been withdrawn as described above, or a fly may be specially dissected for this purpose. In this case the thorax is pierced at the side by one needle so that it can be held, while with the other needle the head is drawn off. The thoracic portion of the salivary glands will be drawn out. The head is then held with one needle while with the other the proboscis is drawn off from the head, the drawn-out portion of the salivary glands remaining attached to the proboscis. In this way a preparation of the proboscis with the thoracic portions of the salivary glands can be obtained very quickly.

When the labium and hypopharynx have been isolated, they are examined for trypanosomes under a cover-glass in saline solution. If it be desired to stain the trypanosomes, the cover-glass is removed after adding

more saline, and the labium and hypopharynx removed to a small drop of fresh serum, in which they are cut with fine needles into eight or ten pieces. The serum is then spread and allowed to dry, after which the film can be stained.

The methods of dissection described above for mosquitoes and tsetse flies can be adapted to the majority of insects.

II. SPIROCHÆTES.

The methods adopted for the study of spirochætes are in many cases the same as those described above for the Protozoa. Certain special methods have been elaborated, and attention may be directed to some of these and to the difficulties which are encountered in the investigation of these organisms.

OBSERVATIONS ON LIVING ORGANISMS.—The study of living spirochætes by transmitted light is very difficult except in the case of *Spirochæta plicatilis, Cristispira balbianii,* and allied forms of large size. For the study of the majority of spirochætes in the living condition dark-ground illumination is essential, but, even with its aid, so narrow and transparent are the organisms that little information regarding internal structure can be obtained. In many cases there is considerable difficulty in distinguishing fibres from true spirochætes, so that not infrequently these structures have been described as living organisms (Fig. 557). Spirochætes can be studied in the fluids in which they occur either in the living condition, or after being killed by osmic vapour, or by the addition of iodine. Sometimes they are rendered more visible by *intra vitam* staining, which may reveal certain irregularities in the structure of the body. It is, however, very difficult to decide whether such irregularities represent the normal structure of the organism or are the result of degenerative changes. It is still more difficult to decide whether granules which occur in the media are stages in the development of spirochætes, as some observers maintain.

Cultivation.—The methods which are employed for the cultivation of spirochætes have been noted above in the section dealing with these organisms. The free-living spirochætes can be grown in the media in which they occur. Those spirochætes which live in the blood or tissues of animals as saprophytes or true parasites will grow in specially prepared media, which consist chiefly of diluted blood-serum, hydrocele, or ascitic fluid from man or animals. They are used in liquid form or in a semi-solid condition produced by the addition of a certain quantity of agar or by coagulation at 100° C. Sometimes the presence in each tube of a small piece of the fresh kidney or other tissue will enable the more delicate spirochætes to grow. The various species of pathogenic *Leptospira* are most readily

cultivated, and they will thrive in Noguchi's serum medium or in the semi-solid blood-agar medium described above for the cultivation of flagellates. The relapsing fever spirochætes are more difficult to maintain, while the organism of syphilis is still more so, though it can be grown indefinitely in serum media containing portions of living tissue.

MAINTENANCE IN LABORATORY ANIMALS.—Various strains of pathogenic spirochætes can be maintained in laboratory animals. The relapsing fever spirochætes of man can readily be inoculated to monkeys, and often to rats and mice. A strain is often more readily passed into rats and mice after it has been inoculated to a monkey. In these animals, as in human beings, recovery is the rule, so that it is necessary to inoculate a fresh animal from the infected one before the spirochætes disappear from the blood. In the same way the chicken spirochæte (*Treponema anserinum* can be maintained by inoculation of chickens or even canaries.

The various pathogenic strains of leptospira can also be kept in laboratory animals, particularly guinea-pigs. Inoculation from animal to animal is best carried out by inoculating intraperitoneally an emulsion of the kidney or liver of an infected animal. From human beings strains can often be isolated by inoculating guinea-pigs intraperitoneally with blood taken at the commencement of the disease or the centrifuged deposit from urine. The naturally occurring rat strains can be obtained by inoculating guinea-pigs with emulsions of the kidneys and liver. It may not be possible to maintain strains indefinitely in guinea-pigs, as after several passages the virulence may be so diminished that infection does not follow inoculation. By a combination of the culture method and animal inoculations strains can be kept virulent for long periods. If kept in culture alone, the virulence to guinea-pigs may be lost after a number of passages.

The spirochætes of syphilis and yaws can be maintained in rabbits by inoculation into the testis or cornea. Similarly, the naturally occurring form in rabbits (*T. cuniculi*) can be handed on from animal to animal.

The spirillum of rat-bite fever can readily be maintained in mice and other laboratory animals by the subcutaneous inoculation of small quantities of blood containing the organisms.

Certain spirochætes which are transmitted from vertebrate to vertebrate by ticks can be maintained in these invertebrates for long periods. The individual ticks (*Ornithodorus* and *Argas*) are very long-lived, and, once infected, retain the infection for the remainder of their lives. Susceptible vertebrates can be infected at any time by allowing the ticks to feed on them or by injecting them with emulsions of crushed ticks. The clear fluid which exudes from the end of a cut leg will often produce infection. Furthermore, the infection passes through the egg, so that the offspring hatched from eggs laid by infective ticks are themselves infective.

PERMANENT PREPARATION OF FIXED AND STAINED SPIROCHÆTES.—
The satisfactory fixation and staining of spirochætes is more difficult
to accomplish than in the case of Protozoa, and even when carried out
satisfactorily little of the internal structure can be detected except in the
case of the larger organisms. In most cases the smaller spirochætes have
been studied in dried films, but it has to be remembered that the drying
process is quite unreliable for giving true pictures of their structure. It
is quite fallacious to make deductions from the appearance of organisms
prepared in this way. Dried films are of use for the detection of the
presence of spirochætes, and in many cases will enable an identification
to be made, but beyond this they are of little value (Fig. 542). Before
any conclusions can be drawn as to their minute structure it is essential
that the organisms be fixed and stained by methods which have been
proved to be accurate for cytological work.

Dried Films.—These are made as described above, and can be stained
with the various modifications of the Romanowsky stain, especially by the
prolonged Giemsa method. Strong solutions of methylene blue, carbol
fuchsin, and other stains will also colour the spirochætes, but in all cases
the staining merely colours the body as a whole, though sometimes
irregularities occur, such as the appearance of granules or bands, but
there are no means of ascertaining that these are not artificially produced
by the drying process.

For the demonstration of spirochætes various silver nitrate methods
are employed. The films are soaked in a solution of silver nitrate and
then placed in a reducing fluid, which causes the deposition of granules of
silver on the bodies of the spirochætes. Apart from causing the organisms
to stand out clearly, and giving some indication of the shape and size of the
body, and the presence or absence of flagella, the method is of little value
for cytological details. The one most commonly used is Fontana's method,
which is carried out as follows:

1. The film is made as thin as possible and dried.

2. The following liquid is then poured on to the film, and renewed
several times during the course of one or two minutes:

Glacial acetic acid	1 part.
Formol	2 parts.
Distilled water	100 ,,

3. After washing for about half a minute in running water, the film is
mordanted by pouring on to it the following liquid and warming gently
over a flame for about half a minute:

Phenol	1 part.
Tannin	5 parts.
Distilled water	100 ,,

4. The film is washed in running water for half a minute and rinsed in distilled water.

5. Ammoniacal silver nitrate solution is poured on the film, which is gently warmed for half a minute. The silver nitrate solution is prepared by adding carefully with a fine pipette a solution of ammonia to a 0·25 per cent. solution of silver nitrate in distilled water till the precipitate which first forms is completely dissolved. A quantity of a silver nitrate solution is then very carefully added with the pipette till the fluid becomes opalescent.

6. The film is then washed, dried, and mounted in Canada balsam.

Indian Ink Method.—This method, which cannot be regarded as a staining method, depends upon the fact that if a thin film of the ink is made on a slide and allowed to dry, the ink forms a continuous layer except where minute objects occur. If a drop of fluid containing spirochætes be mixed with a drop of ink and a film made, after drying it will be found that the spirochætes are free from the deposit, so that the general shape of the body is represented by a clear area in the dark background. The method, which is useful for the detection of spirochætes, affords no information regarding the structure of the organisms apart from the arrangement of the spiral turns and shape and size of the body.

Wet Fixed Films.—It is probable that this method of preparation, which is carried out as described above for the Protozoa, gives the most accurate picture of the structure of spirochætes. The films can be fixed in any of the fixatives mentioned, but the staining is more difficult to carry out. The best methods are probably the long Giemsa method, as described for wet fixed films, and the iron hæmatoxylin method, which often give good results.

Section Cutting.—The staining of spirochætes in sections is still more difficult to accomplish than in films. Tissues fixed in any of the fixatives described above may be stained by the long Giemsa method, which will sometimes stain the spirochætes. The best method, however, for the demonstration of spirochætes in tissues is the silver nitrate method of Levaditi, or one of its modifications, which are adaptations of that of Cajal for staining nerve fibrils. Levaditi's method is carried out as follows:

1. Small pieces of tissue are fixed for twenty-four hours in a 10 per cent. solution of formalin.

2. Transfer to 95 per cent. spirit for twenty-four hours.

3. Wash in distilled water for a few minutes.

4. Soak in a 1·5 per cent. solution of silver nitrate for three days at a temperature of about 38° C.

5. Place in the following reducing fluid for twenty-four hours:

Pyrogallic acid	..	..	..	..	..	4 grams.
Formol ..	..	..	..	..	..	5 c.c.
Distilled water	..	..	..	..	..	100 c.c.

6. The tissue is then washed in distilled water, dehydrated, cleared, and embedded in paraffin in the usual manner.

Levaditi's method has been improved by Dobell, whose technique has the advantage of eliminating, to a large extent, the staining of the tissues by the silver so that the spirochætes stand out in greater contrast, while subsequent staining of the tissues is more satisfactory.

1. Fix in 10 per cent. solution of formalin for one to three days.

2. Transfer to 95 per cent. spirit or absolute alcohol for twenty-four hours.

3. Transfer through graded alcohols to distilled water till tissue is free from all traces of alcohol.

4. Soak in 0·25 to 0·5 per cent. solution of silver nitrate for eighteen to twenty-four hours in the dark at 37° C. (incubator).

5. Wash in several changes of distilled water for an hour till washings are free from silver nitrate.

6. Immerse in 0·5 to 1 per cent. solution of hydroquinone in 50 per cent. alcohol for eighteen to twenty-four hours at room temperature.

7. Wash in 70 per cent. alcohol, dehydrate and embed in paraffin in usual manner.

The sections, after fixing on slides, are simply washed in xylol to remove the paraffin, and mounted in Canada balsam, or they may be taken into water and stained by any of the ordinary stains before mounting. The spirochætes stand out as dark filaments, owing to the deposit upon them of silver, as in films stained by Fontana's method. The method is very largely used for the detection of *T. pallidum* and species of *Leptospira* in tissues (Figs. 552 and 560), and has been applied to the relapsing fever spirochætes in lice (Fig. 545).

RULES OF NOMENCLATURE

Zoological nomenclature in its present form commenced with the binomial system published by Linnæus in the tenth edition of his *Systema Naturæ* in 1758. After this, various proposals and regulations were drawn up in an attempt to clear away the confusion, but not having international recognition, they met with little success. Finally, the International Congress of Zoology, held at Cambridge in 1898, set up an International Commission on Zoological Nomenclature as a permanent body. They issued a Code of Rules, which, together with the Recommendations under the Articles, the Appendix to the Code, and the Opinions of the Commission given from time to time, control and regulate the whole question of the naming of animals. Not infrequently the application of the Rules may appear to lead to disorder, as, for instance, when a name which has been long in use has to be discarded for one which has been overlooked or forgotten. This is, however, of only a temporary nature, while the number of instances in which it occurs is negligible when compared with the confusion which would inevitably result if no such guiding rules were strictly adhered to. For the convenience of those who may not be able to have access to the Rules, those published in the *Proceedings of the Ninth International Congress of Zoology*, held at Monaco in 1913, are reproduced here.

INTERNATIONAL RULES OF ZOOLOGICAL NOMENCLATURE.
RULES AND RECOMMENDATIONS.
General Considerations.

ARTICLE 1. — Zoological nomenclature is independent of botanical nomenclature in the sense that the name of an animal is not to be rejected simply because it is identical with the name of a plant. If, however, an organism is transferred from the vegetable to the animal kingdom its botanical names are to be accepted in zoological nomenclature with their original botanical status; and if an organism is transferred from the animal to the vegetable kingdom its names retain their zoological status.

RECOMMENDATION. — It is well to avoid introducing into zoology as generic names such names as are in use in botany.

ARTICLE 2. — The scientific designation of animals is uninominal for subgenera and all higher groups, binominal for species, and trinominal for subspecies.

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ARTICLE 3. — The scientific names of animals must be words which are either Latin or Latinized, or considered and treated as such in case they are not of classic origin.

Family and Subfamily Names.

ARTICLE 4. — The name of a family is formed by adding the ending *idae*, the name of a subfamily by adding *inae*, to the stem of the name of its type genus.

ARTICLE 5. — The name of a family or subfamily is to be changed when the name of its type genus is changed.

Generic and Subgeneric Names.

ARTICLE 6. — Generic and subgeneric names are subject to the same rules and recommendations, and from a nomenclatural standpoint they are co-ordinate—that is, they are of the same value.

ARTICLE 7. — A generic name becomes a subgeneric name when the genus so named becomes a subgenus, and *vice versa*.

ARTICLE 8. — A generic name must consist of a single word, simple or compound, written with a capital initial letter, and employed as a substantive in the nominative singular. Examples: *Canis, Perca, Ceratodus, Hymenolepis*.

RECOMMENDATION. — Certain biological groups which have been proposed distinctly as collective groups, not as systematic units, may be treated for convenience as if they were genera, but they require no type species. Examples: *Agamodistomum, Amphistomulum, Agamofilaria, Agamomermis, Sparganum*.

RECOMMENDATIONS. — The following words may be taken as generic names:

a) Greek substantives, for which the rules of Latin transcription [transliteration (see Appendix F)] should be followed. Examples: *Ancylus, Amphibola, Aplysia, Pompholyx, Physa, Cylichna*.

b) Compound Greek words, in which the attributive should precede the principal word. Examples: *Stenogyra, Pleurobranchus, Tylodina, Cyclostomum, Sarcocystis, Pelodytes, Hydrophilus, Rhizobius*.

This does not, however, exclude words formed on the model of *Hippopotamus*—namely, words in which the attributive follows the principal word. Examples: *Philydrus, Biorhiza*.

c) Latin substantives. Examples: *Ancilla, Auricula, Dolium, Harpa, Oliva*. Adjectives (*Prasina*) and past participles (*Productus*) are not recommended.

d) Compound Latin words. Examples: *Stiliger, Dolabrifer, Semifusus*.

e) Greek or Latin derivatives expressing diminution, comparison, resemblance, or possession. Examples: *Dolium, Doliolum ; Strongylus, Eustrongylus ; Limax, Limacella, Limacia, Limacina, Limacites, Limacula ; Lingula, Lingulella, Lingulepis, Lingulina, Lingulops, Lingulopsis ; Neomenia, Proneomenia ; Buteo, Archibuteo ; Gordius, Paragordius, Polygordius.*

f) Mythological or heroic names. Examples: *Osiris, Venus, Brisinga, Velleda, Crimora.* If not Latin, these should be given a Latin termination (*Aegirus, Göndulia*).

g) Proper names used by the ancients. Examples: *Cleopatra, Belisarius, Melania.*

h) Modern patronymics, to which is added an ending to denote dedication.

a. Names terminating with a consonant take the ending *ius, ia,* or *ium.* Examples: *Selysius, Lamarckia, Köllikeria, Mülleria, Stålia, Krøyeria, Ibañezia.*

β. Names terminating with the vowels *e, i, o, u,* or *y* take the ending *us, a,* or *um.* Examples: *Blainvillea, Wyvillea, Cavolinia, Fatioa, Bernaya, Quoya, Schulzea.*

γ. Names terminating with *a* take the ending *ia.* Example: *Danaia.*

δ. In generic names formed from patronymics, the particles are omitted if not coalesced with the name, but the articles are retained. Examples: *Blainvillea, Benedenia, Chiajea, Lacepedea, Dumerilia.*

ε. With patronymics consisting of two words, only one of these is used in the formation of a generic name. Examples: *Selysius, Targionia, Edwardsia, Duthiersia.*

ζ. The use of proper names in the formation of compound generic names is objectionable. Examples: *Eugrimmia, Buchiceras, Heromorpha, Möbiusispongia.*

i) Names of ships, which should be treated the same as mythological names (*Vega*) or as modern patronymics. Examples: *Blakea, Hirondellea, Challengeria.*

j) Barbarous names, that is, words of non-classic origin. Examples: *Vanikoro, Chilosa.* Such words may receive a Latin termination. Examples: *Yetus, Fossarus.*

k) Words formed by an arbitrary combination of letters. Examples: *Neda, Clanculus, Salifa, Torix.*

l) Names formed by anagram. Examples: *Dacelo, Verlusia, Linospa.*

ARTICLE 9. — If a genus is divided into subgenera, the name of the typical subgenus must be the same as the name of the genus (see Art. 25).

ARTICLE 10. — When it is desired to cite the name of a subgenus, this name is to be placed in parentheses between the generic and the specific names. Examples: *Vanessa (Pyrameis) cardui.*

Specific and Subspecific Names.

ARTICLE 11. — Specific and subspecific names are subject to the same rules and recommendations, and from a nomenclatural standpoint they are co-ordinate—that is, they are of the same value.

ARTICLE 12. — A specific name becomes a subspecific name when the species so named becomes a subspecies, and *vice versa.*

ARTICLE 13. — While specific substantive names derived from names of persons may be written with a capital initial letter, all other specific names are to be written with a small initial letter. Examples: *Rhizostoma Cuvieri* or *Rh. Cuvieri, Francolinus Lucani* or *F. lucani, Hypoderma Diana* or *H. diana, Laophonte Mohammed* or *L. mohammed, Œstrus ovis, Corvus corax.*

ARTICLE 14. — Specific names are:

a) Adjectives, which must agree grammatically with the generic name. Example: *Felis marmorata.*

b) Substantives in the nominative in apposition with the generic name. Example: *Felis leo.*

c) Substantives in the genitive. Examples: *Rosae, sturionis, antillarum, galliae, sancti-pauli, sanctae-helenae.*

If the name is given as a dedication to one or several persons, the genitive is formed in accordance with the rules of Latin declination in case the name was employed and declined in Latin. Examples: *Plinii, Aristotelis, Victoris, Antonii, Elisabethae, Petri* (given name).

If the name is a modern patronymic, the genitive is always formed by adding to the exact and complete name an *i* if the person is a man, or an *ae* if the person is a woman, even if the name has a Latin form; it is placed in the plural if the dedication involves several persons of the same name. Examples: *Cuvieri, Möbiusi, Nuñezi, Merianae, Sarasinorum, Bosi* (not *Bovis*), *Salmoni* (not *Salmonis*).

RECOMMENDATION. — The best specific name is a Latin adjective, short, euphonic, and of easy pronunciation. Latinized Greek words or barbarous words may, however, be used. Examples: *gymnocephalus, echinococcus, ziczac, aguti, hoactli, urubitinga.*

It is well to avoid the introduction of the names *typicus* and *typus* as new names for species or subspecies, since these names are always liable to result in later confusion.

ARTICLE 15. — The use of compound proper names indicating dedication, or of compound words indicating a comparison with a simple object, does not form an exception to Art. 2. In these cases the two words composing the specific name are written as one word, with or

without the hyphen. Examples: *Sanctae-catharinae* or *sanctaecatharinae*, *jan-mayeni* or *janmayeni*, *cornu-pastoris* or *cornupastoris*, *cor-anguinum* or *coranguinum*, *cedo-nulli* or *cedonulli*.

Expressions like *rudis planusque* are not admissible as specific names.

ARTICLE 16. — Geographic names are to be given as substantives in the genitive, or are to be placed in an adjectival form. Examples: *sancti-pauli, sanctae-helenae, edwardiensis, diemenensis, magellanicus, burdigalensis, vindobonensis.*

RECOMMENDATION. — Geographic names used by the Romans or by Latin writers of the middle ages are to be adopted in preference to more recent forms. Words like *bordeausiacus* and *viennensis* are poor, but are not to be rejected on this account.

ARTICLE 17. — If it is desired to cite the subspecific name, such name is written immediately following the specific name, without the interposition of any mark of punctuation. Example: *Rana esculenta marmorata* Hallowell, but not *Rana esculenta (marmorata)* or *Rana marmorata* Hallowell.

ARTICLE 18. — The notation of hybrids may be given in several ways; in all cases the name of the male parent precedes that of the female parent, with or without the sexual signs:

a) The names of the two parents are united by the sign of multiplication ($\times$). Examples: *Capra hircus* ♂ $\times$ *Ovis aries* ♀ and *Capra hircus* $\times$ *Ovis aries* are equally good formulæ.

b) Hybrids may also be cited in form of a fraction, the male parent forming the numerator and the female parent the denominator. Example: $\dfrac{\textit{Capra hircus}}{\textit{Ovis aries}}$. This second method is in so far preferable that it permits the citation of the person who first published the hybrid form as such. Example: $\dfrac{\textit{Bernicla canadensis}}{\textit{Anser cygnoides}}$ Rabé.

c) The fractional form is also preferable in case one of the parents is itself a hybrid. Example: $\dfrac{\textit{Tetrao tetrix} \times \textit{Tetrao urogallus}}{\textit{Gallus gallus}}$. In the latter case, however, the parentheses may be used. Example: (*Tetrao tetrix* $\times$ *Tetrao urogallus*) $\times$ *Gallus gallus*.

d) When the parents of the hybrid are not known as such (parents), the hybrid takes provisionally a specific name, the same as if it were a true species, namely, as if it were not a hybrid; but the generic name is preceded by the sign of multiplication. Example: $\times$ *Coregonus dolosus* Fatio.

Formation, Derivation, and Orthography of Zoological Names.

ARTICLE 19. — The original orthography of a name is to be preserved unless an error of transcription, a *lapsus calami*, or a typographical error is evident.

RECOMMENDATION. — For scientific names it is advisable to use some other type than that used for the text. Example: *Rana esculenta* Linné, 1758, lives in Europe.

ARTICLE 20. — In forming names derived from languages in which the Latin alphabet is used, the exact original spelling, including diacritic marks, is to be retained. Examples: *Selysius, Lamarckia, Köllikeria, Mülleria, Stålia, Krøyeria, Ibañezia, Möbiusi, Medići, Čžjžeki, spitzbergensis, islandicus, paraguayensis, patagonicus, barbadensis, faröensis.*

RECOMMENDATIONS. — The prefixes *sub* and *pseudo* should be used only with adjectives and substantives, *sub* with Latin words, *pseudo* with Greek words, and they should not be used in combination with proper names. Examples: *subviridis, subchelatus, Pseudacanthus, Pseudophis, Pseudomys.* Words like *sub-wilsoni* and *pseudo-grateloupana* are not recommended.

The terminations *oides* and *ides* should be used in combination only with Greek or Latin substantives; they should not be used in combination with proper names. •

Geographic and patronymic names from countries which have no recognized orthography or which do not use the Latin alphabet should be transcribed into Latin according to the rules adopted by the Geographic Society of Paris (See Appendix G).

In proposing new names based upon personal names, which are written sometimes with ä, ö, or ü, at other times with ae, oe, and ue, it is recommended that authors adopt ae, oe, and ue. Example: *muelleri* in preference to *mülleri*.

Author's Name.

ARTICLE 21. — The author of a scientific name is that person who first publishes the name in connection with an indication, a definition, or a description, unless it is clear from the contents of the publication that some other person is responsible for said name and its indication, definition, or description.

ARTICLE 22. — If it is desired to cite the author's name, this should follow the scientific name without interposition of any mark of punctuation; if other citations are desirable (date, *sp. n., emend., sensu stricto,* etc.), these follow after the author's name, but are separated from it by a comma or by parenthesis. Examples: *Primates* Linné, 1758, or *Primates* Linné (1758).

RECOMMENDATION. — When the name of the author of a scientific name is abbreviated, the writer will do well to conform to the list of abbreviations published by the Zoological Museum of Berlin.

ARTICLE 23. — When a species is transferred to another than the original genus or the specific name is combined with any other generic name than that with which it was originally published, the name of the author of the specific name is retained in the notation, but placed in parentheses. Examples: *Taenia lata* Linné, 1758, and *Dibothriocephalus latus* (Linné, 1758); *Fasciola hepatica* Linné, 1758, and *Distoma hepaticum* (Linné, 1758).

If it is desired to cite the author of the new combination, his name follows the parentheses. Example: *Limnatis nilotica* (Savigny, 1820) Moquin-Tandon, 1826.

ARTICLE 24. — When a species is divided, the restricted species to which the original specific name of the primitive species is attributed may receive a notation indicating both the name of the original author and the name of the reviser. Example: *Taenia solium* Linné, partim, Goeze.

The Law of Priority.

ARTICLE 25. — The valid name of a genus or species can be only that name under which it was first designated on the condition:

a) That this name was published and accompanied by an indication, or a definition, or a description; and

b) That the author has applied the principles of binary nomenclature.

Application of the Law of Priority.

ARTICLE 26. — The tenth edition of Linné's *Systema Naturae*, 1758, is the work which inaugurated the consistent general application of the binary nomenclature in zoology. The date 1758, therefore, is accepted as the starting-point of zoological nomenclature and of the Law of Priority.

ARTICLE 27. — The Law of Priority obtains, and consequently the oldest available name is retained:

a) When any part of an animal is named before the animal itself;

b) When any stage in the life-history is named before the adult;

c) When the two sexes of an animal have been considered as distinct species or even as belonging to distinct genera;

d) When an animal represents a regular succession of dissimilar generations which have been considered as belonging to different species or even to different genera.

ARTICLE 28. — A genus formed by the union of two or more genera or subgenera takes the oldest valid generic or subgeneric name of its components. If the names are of the same date, that selected by the first reviser shall stand.

The same rule obtains when two or more species or subspecies are united to form a single species or subspecies.

RECOMMENDATION. — In absence of any previous revision, the establishment of precedence by the following method is recommended:

a) A generic name accompanied by specification of a type has precedence over a name without such specification. If all or none of the genera have types specified, that generic name takes precedence the diagnosis of which is most pertinent.

b) A specific name accompanied by both description and figure stands in preference to one accompanied only by a diagnosis or only by a figure.

c) Other things being equal, that name is to be preferred which stands first in the publication (page precedence).

ARTICLE 29. — If a genus is divided into two or more restricted genera, its valid name must be retained for one of the restricted genera. If a type was originally established for said genus, the generic name is retained for the restricted genus containing said type.

RECOMMENDATION. — To facilitate reference, it is recommended that when an older species is taken as type of a new genus, its name should be actually combined with the new generic name in addition to citing it with the old generic name. Example: *Gilbertella* Eigenmann, 1903, Smithsonian Misc. Coll., v. 45, p. 147, type *Gilbertella alata* (Steindachner)= *Anacyrtus alatus* Steindachner.

ARTICLE 30. — The designation of type species of genera shall be governed by the following rules (*a-g*), applied in the following order of precedence:

I. Cases in which the generic type is accepted *solely* upon the basis of the original publication:

a) When in the original publication of a genus one of the species is definitely designated as type, this species shall be accepted as type, regardless of any other considerations. (Type by original designation.)

b) If in the original publication of a genus, *typicus* or *typus* is used as a *new* specific name for one of the species, such use shall be construed as " type by original designation."

c) A genus proposed with a single original species takes that species as its type. (Monotypical genera.)

d) If a genus, without originally designated (see *a*) or indicated (see *b*) type, contains among its original species one possessing the generic name as its specific or subspecific name, either as valid name or synonym,

that species or subspecies becomes *ipso facto* type of the genus.　(Type by absolute tautonymy.)

II. Cases in which the generic type is accepted not solely upon basis of the original publication:

e) The following species are excluded from consideration in determining the types of genera:

a. Species which were not included under the generic name at the time of its original publication.

β. Species which were *species inquirendae* from the standpoint of the author of the generic name at the time of its publication.

γ. Species which the author of the genus doubtfully referred to it.

f) In case a generic name without originally designated type is proposed as a substitute for another generic name, with or without type, the type of either, when established, becomes *ipso facto* type of other.

g) If an author, in publishing a genus with more than one valid species, fails to designate (see *a*) or to indicate (see *b*, *d*) its type, any subsequent author may select the type, and such designation is not subject to change. (Type by subsequent designation.)

The meaning of the expression " select the type " is to be rigidly construed.　Mention of a species as an illustration or example of a genus does not constitute a selection of a type.

III. RECOMMENDATIONS. — In selecting types by subsequent designation, authors will do well to govern themselves by the following recommendations:

h) In case of Linnaean genera, select as type the most common or the medicinal species.　(Linnaean rule, 1751).

i) If a genus, without designated type, contains among its original species one possessing as a specific or subspecific name, either as valid name or synonym, a name which is virtually the same as the generic name, or of the same origin or same meaning, preference should be shown to that species in designating the type, unless such preference is strongly contra-indicated by other factors.　(Type by virtual tautonymy.)　Examples: *Bos taurus*, *Equus caballus*, *Ovis aries*, *Scomber scombrus*, *Sphaerostoma globiporum*; contra-indicated in *Dipetalonema* (compare species *Filaria dipetala*, of which only one sex was described, based upon one specimen and not studied in detail).

j) If the genus contains both exotic and non-exotic species from the standpoint of the original author, the type should be selected from the non-exotic species.

k) If some of the original species have later been classified in other genera, preference should be shown to the species still remaining in the original genus.　(Type by elimination.)

l) Species based upon sexually mature specimens should take precedence over species based upon larval or immature forms.

m) Show preference to species bearing the name *communis*, *vulgaris*, *medicinalis* or *officinalis*.

n) Show preference to the best described, best figured, best known, or most easily obtainable species, or to one of which a type specimen can be obtained.

o) Show preference to a species which belongs to a group containing as large a number of the species as possible.   (De Candolle's rule.)

p) In parasitic genera, select, if possible, a species which occurs in the man or some food animal, or in some very common and widespread host species.

q) All other things being equal, show preference to a species which the author of the genus actually studied at or before the time he proposed the genus.

r) In case of writers who habitually placed a certain leading or typical species first as " chef de file," the others being described by comparative reference to this, this fact should be considered in the choice of the type species.

s) In case of those authors who have adopted the " first species rule " in fixing generic types, the first species named by them should be taken as types of their genera.

t) All other things being equal, page precedence should obtain in selecting a type.

ARTICLE 31. — The division of a species into two or more restricted species is subject to the same rules as the division of a genus.   But a specific name which undoubtedly rests upon an error of identification cannot be retained for the misdetermined species even if the species in question are afterwards placed in different genera.   Example: *Taenia pectinata* Goeze, 1782=*Cittotaenia pectinata* (Goeze), but the species erroneously determined by Zeder, 1800, as " *Taenia pectinata* Goeze "= *Andrya rhopalocephala* (Rehm); the latter species does not take the name *Andrya pectinata* (Zeder).

Rejection of Names.

ARTICLE 32. — A generic or a specific name, once published, cannot be rejected, even by its author, because of inappropriateness.   Examples: Names like *Polyodon*, *Apus*, *albus*, etc., when once published, are not to be rejected because of a claim that they indicate characters contradictory to those possessed by the animals in question.

ARTICLE 33. — A name is not to be rejected because of tautonymy— that is, because the specific or the specific and subspecific names are

identical with the generic name.　Examples: *Trutta trutta, Apus apus apus.*

ARTICLE 34. — A generic name is to be rejected as a homonym when it has previously been used for some other genus of animals.　Example: *Trichina* Owen, 1835, nematode, is rejected as homonym of *Trichina* Meigen, 1830, insect.

ARTICLE 35. — A specific name is to be rejected as a homonym when it has previously been used for some other species of the same genus. Examples: *Taenia ovilla* Rivolta, 1878 (n. sp.) is rejected as homonym of *T. ovilla* Gmelin, 1790.

When in consequence of the union of two genera, two different animals having the same specific or subspecific name are brought into one genus, the more recent specific or subspecific name is to be rejected as a homonym.

Specific names of the same origin and meaning shall be considered homonyms if they are distinguished from each other only by the following differences:

a) The use of *ae, oe,* and *e,* as *caeruleus, coeruleus, ceruleus ; ei, i,* and *y,* as *chiropus, cheiropus ; c* and *k,* as *microdon, mikrodon.*

b) The aspiration or non-aspiration of a consonant, as *oxyryncus, oxyrhynchus.*

c) The presence or absence of a *c* before *t,* as *autumnalis, auctumnalis.*

d) By a single or double consonant: *litoralis, littoralis.*

e) By the endings *ensis* and *iensis* to a geographical name, as *timorensis, timoriensis.*

ARTICLE 36. — Rejected homonyms can never be again used. Rejected synonyms can again be used in case of the restoration of erroneously suppressed groups.　Example: *Taenia Giardi* Moniez, 1879, was suppressed as a synonym of *Taenia ovilla* Rivolta, 1878; later it was discovered that *Taenia ovilla* was preoccupied (*Taenia ovilla* Gmelin, 1790). *Taenia ovilla,* 1878, is suppressed as a homonym, and can never again be used; it was stillborn and cannot be brought to life, even when the species is placed in another genus (*Thysanosoma*).　*Taenia Giardi,* 1879, which was suppressed as a synonym, becomes valid upon the suppression of the homonym *Taenia ovilla* Rivolta.

RECOMMENDATIONS. — It is well to avoid the introduction of new generic names which differ from generic names already in use only in termination or in a slight variation in spelling which might lead to confusion.　But when once introduced, such names are not to be rejected on this account.　Examples: *Picus, Pica ; Polyodus, Polyodon, Polyodonta, Polyodontas, Polyodontus.*

The same recommendation applies to new specific names in any given

genus. Examples: *necator, necatrix ; furcigera, furcifera ; rhopalocephala, rhopaliocephala.*

If from the radical of a geographic name two or more adjectives are derived, it is not advisable to use more than one of them as specific name in the same genus, but if once introduced, they are not to be rejected on this account. Examples: *hispanus, hispanicus ; moluccensis, moluccanus ; sinensis, sinicus, chinensis ; ceylonicus, zeylanicus.*

The same recommendation applies also to other words derived from the same radical and differing from each other only in termination or by a simple change in spelling.

APPENDIX.

A. — It is very desirable that the proposition of every new systematic group should be accompanied by a diagnosis, both individual and differential, of said group in English, French, German, Italian, or Latin. This diagnosis should state in what museum the type specimen has been deposited, and should give the museum number of said specimen.

It is recommended that in published descriptions of a new species or new subspecies, only one specimen be designated and labelled as *type*, the other specimens examined by the author at the same time being *paratypes*.

B. — In publications issued in any other language than English, French, German, Italian, or Latin, it is very desirable that the explanation of figures be translated into one of these tongues.

C. — The metric system of weights and measures and the centigrade thermometer of Celsius are adopted as standard. The *micron* (0·001 mm.), represented by the Greek letter μ, is adopted as the unit of measure in microscopic work.

D. — The indication of enlargement or of reduction, which is very desirable for the comprehension of an illustration, should be expressed in figures rather than by mentioning the system of lenses used.

E. — The indication of enlargement or reduction of an object is usually linear. The sign of multiplication is used for enlargement, and the fraction for reduction. Examples: $\times 50$ indicates that the object is enlarged 50 times; $\frac{1}{50}$ indicates that it is reduced to $\frac{1}{50}$th.

If it is desired to specify that the enlargement is linear, surface, or mass, this may be done as follows: $\times 50^1$ indicates linear enlargement; $\times 50^2$ indicates surface enlargement; $\times 50^3$ indicates mass enlargement.

F. — *Transliteration of Greek Words*. — The following table indicates the manner in which Greek words should be transliterated:

ε = e	(ὑάλεος)	— Hyalea, *not* Hyalaea
η = e	(πειρήνη)	— Pirena, *not* Pirina
final η = a	(πειρήνη)	— Pirena, *not* Pirene
θ = th	(τηθύς)	— Tethys, *not* Tetys
ι = i	(βαλίος)	— Balia, *not* Balea
κ = c	(ἱπποκρήνη)	— Hippocrena, *not* Hippochrenes
ξ = x	(ξένος)	— Xenus, Xenophora
ρ = r	(πτερόν)	— Pterum
υ = y	(ὑβός)	— Hybolithus, *not* Hibolites
αι = ae	(λιμναῖος)	— Limnaea, *not* Limnea
αυ = au	(γλαυκός)	— Glaucus
ει = i	(χεῖλος)	— Chilostomum, *not* Cheilostoma
ευ = eu	(εὖρος)	— Eurus
ῳ, οι = oe	(οἰκέω)	— Dioeca, Dendroeca, *not* Dioica, Dendroica
final ον = um	(ἐφίππιον)	— Ephippium, *not* Ephippion
final ος = us	(ὀμφαλός)	— Euomphalus, *not* Euomphalos
ου = u	(λουτήριον)	— Luterium, *not* Lotorium
γγ = ng	(ἀγγαρεία)	— Angaria
γχ = nch	(ἀγχιστομον)	— Anchistomum, *not* Angistoma
γκ = nc	(ἄνκιστρον)	— Ancistrodon, *not* Agkistrodon
ῥ = rh	(ῥέα)	— Rhea
ἑ = he	(ἑρμαία)	— Hermaea, *not* Ermaea

G. — *Transcription of geographic and proper names*. — The geographic names of nations which employ the Latin characters are to be written with the orthography of the country in which they originate.

The following paragraphs apply only to the geographic names of countries which have no true alphabet or which use letters that are different from the Latin alphabet.

Names of places, however, which have been established by long usage preserve their usual orthography. Examples: *Alger, Moscow*.

1. The vowels *a, e, i*, and *o* are pronounced as in French, Italian, Spanish, or German. The letter *e* is never mute.

2. The French sound *u* is represented by *ü*, with dieresis, as in German.

3. The French sound *ou* is represented by *u*, as in Italian, Spanish, German, etc.

4. The French sound *eu* is represented by *œ*, pronounced as in the French word *œil*.

5. The long sound of a vowel is indicated by circumflex accent; the interrupted sound is indicated by an apostrophe.

6. The consonants *b, d, f, j, k, l, m, n, p, q, r, t, v*, and *z* are pronounced as in French.

7. The letters *g* and *s* always have the hard sound, as in the French words *gamelle* and *sirop*.

8. The sound represented in French by *ch* is designated by *sh*. Examples: *Shérif, Kashgar*.

9. *Kh* represents the harsh guttural; *gh* represents the soft guttural of the Arabs.

10. *Th* represents the sound which terminates the English word *path* (θ in Greek). *Dh* represents the sound which commences with the English word *those* (δ in Greek).

11. Aside from such employment (9, 10) of the letter *h* modifying the letter which precedes it, *h* is always aspirated; the apostrophe is therefore never used before a word commencing with *h*.

12. The semivowel represented by *y* is pronounced as in *yole*.

13. The semivowel *w* is pronounced as in the English word *William*.

14. The double sounds *dj*, *tch*, *ts*, etc., are indicated by letters representing the sounds which compose them. Example: Matshim.

15. The *ñ* is pronounced *gn*, as in *seigneur*.

16. The letters *x*, *c*, and *q* are not used, since they are duplicates of other letters representing the same sounds; but *q* may serve to indicate the Arabic *qaf*, and the soft aspirate may be used to represent the Arabic *ain*.

An attempt should be made to indicate as exactly as possible, by means of the letters given above, the local pronunciation without trying to give a complete representation of all the sounds which are heard.

PART V

BLOOD PARASITES OF VERTEBRATES AND TRYPANOSOMIDÆ OF INVERTEBRATES

BLOOD PARASITES OF VERTEBRATES AND TRYPANOSOMIDÆ OF INVERTEBRATES

THE following lists of hosts are divided into two sections. The first contains the vertebrate hosts of blood parasites and the second the invertebrate hosts of Trypanosomidæ belonging to the genera *Leptomonas, Crithidia*, and *Herpetomonas*. In many cases the recorded host name does not appear to be the correct one, and in such cases it is placed in brackets, the correct name being given without brackets. The parasites occurring in any host are given under the host name used by the recorder, so that in cases where correct and incorrect names are found reference to both will have to be made for the complete list of parasites. In all cases the name of the recorder, the year, and locality have been given.

I. VERTEBRATE HOSTS OF BLOOD PARASITES.

The following abbreviations are used: *B.* = *Babesia*; *H.* = *Hæmoproteus*; *Hg.* = *Hæmogregarina*; *Hp.* = *Hepatozoon*; *L.* = *Leucocytozoon*; *P.* = *Plasmodium*; *T.* = *Trypanosoma*; *Tp.* = *Trypanoplasma*; *Tx.* = *Toxoplasma*. It is necessary to point out that the records under the name of Plimmer refer to animals which had died in the Zoological Society's Gardens in London. Similarly, those marked Z.S., 1925, refer to a series of blood-films made from animals which had died in the Zoological Gardens by Dr. H. H. Scott, Pathologist to the Zoological Society, and which were examined by him and the writer. The records, which occur in Dr. Scott's reports, will probably be published in the *Proceedings of the Zoological Society* during 1926.

In many cases there has been considerable difficulty in the identification of the hosts from the names given by recorders, and in some cases this has been quite impossible. Much assistance has been given by specialists at the British Museum (National History) who have enabled the names to be checked from their lists of Mammalia, those of birds based on R. Bowdler Sharpe's Hand List of the Genera and Species of Birds, those of the Reptilia and Amphibia based on Boulenger's Catalogue, and those of fish based on Gunther's Catalogue.

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MAMMALIA.

Acomys sp. (spiny mouse): *T. acomys*, Wenyon, 1909, Sudan.

Æpyceros melampus (mpala): *T. brucei*, Kinghorn and Yorke, 1912, Rhodesia. *T. congolense*, Bruce *et al.*, 1914, Nyasaland; Kinghorn and Yorke, 1912, Rhodesia. *T. capræ*, Bruce *et al.*, 1914, Nyasaland. *B.* sp., Ross, P. H., 1911, East Africa.

Æthechinus algirus =(Erinaceus algirus).

Akodon fuliginosus (vole mouse): *T. akodoni*, Carini and Maciel, 1915, Brazil. *Hp. akodoni*, Carini and Maciel, 1915, Brazil.

Alouatta senicula =(Mycetes seniculus) (howling monkey): *T.* sp., Brimont, 1909, Guiana.

Alouatta ursina =(Mycetes ursinus).

Anomalurus fraseri (spiny-tailed flying squirrel): *T. denysi*, Rodhain, Pons, Vandenbranden and Bequaert, 1912, Congo.

(Anthropopithecus gorilla) =Gorilla gorilla (gorilla): *T. lewisi* var. *primatum*, Reichenow, 1917, 1920, Cameroons. *P. vivax*, Reichenow, 1920, Cameroons. *P. falciparum*, Reichenow, 1920, Cameroons.

Anthropopithecus troglodytes (chimpanzee): *T. lewisi* var. *primatum*, Reichenow, 1917, 1920, Cameroons; Ziemann, 1902, Cameroons. *P. kochi*, Lühe, 1906, Cameroons. *P. falciparum*, Reichenow, 1920, Cameroons. *P. vivax*, Reichenow, 1920, Cameroons. *P. malariæ*, Reichenow, 1920, Cameroons. *P.* sp. (?) *falciparum*, Blacklock and Adler, 1922, Sierra Leone; Adler, 1923, Sierra Leone.

Apodemus agrarius =(Mus agrarius).

Apodemus sylvaticus =(Mus sylvaticus).

(Arctomys marmota) =Marmota marmota (marmot): *Hp. plicata marmotæ*, Martoglio, 1913, Somaliland.

Arlibeus perspicillatus =Phyllostoma perspicillatum.

(Arvicanthis barbarus pulchellus) =Lemniscomys barbarus pulchellus (striped mouse): *T. arvicanthi*, Delanoë, 1915, West Africa.

(Arvicanthis pumilio) =Rhabdomys pumilio (striped mouse): *T. arvicanthi*, Delanoë, 1915, West Africa; *T. lewisi*, Fantham, 1920, South Africa.

(Arvicanthis zebra) =Lemniscomys zebra (striped mouse): *T. avicularis*, Wenyon, 1909, Sudan. *B. avicularis*, Wenyon, 1909, Sudan.

Arvicola amphibius =(Microtus amphibius).

Arvicola arvalis =(Microtus arvalis) (field vole): *Hg. arvalis*, Martoglio, 1913, Somaliland.

Ateles pentadactylus (spider monkey): *T. lesourdi*, Leger and Porry, 1918, French Guiana.

Ateles sp. (spider monkey): *P.* sp., Scidelin, 1912, Yucatan.

Atlantoxerus getulus (Barbary ground squirrel): *Hp. getulum*, Sergent, 1921, North Africa.

Axis axis =(Cervus axis).

Bear (Ursus sp.): *P.*, Yakimoff, Kohl-Yakimoff and Korssak, 1910, Russia.

Bos brachycerus (Angola buffalo): *B. brachyceri*, de Mello and Rebello, 1923, Angola.

Boselaphus tragocamelus (nylghai): *B.* sp., Lichtenheld, 1910, East Africa.

(Brachyuus calva) =Ouakaria calva (ouakari monkey): *P. brasilianum* and *T. prowazeki*, Gonder and Berenberg-Gossler, 1908, South America.

Bradypus tridactylus (three-toed sloth): *B. brimonti*, Leger and Mouzels, 1917, French Guiana.

Bubalis cokei : *B.* sp., Ross, P. H., 1911, East Africa.

Bubalis lichtensteini (hartebeest): *T. brucei*, Bruce *et al.*, 1913, Nyasaland; Taute, 1913, Tanganyika; Kinghorn and Yorke, 1912, Rhodesia. *T. congolense*, Bruce *et al.*, 1913, Nyasaland; Kleine and Fischer, 1911, East Africa. *T.* sp., Montgomery and Kinghorn, 1908, Rhodesia.

Buffalo (Bubalus): *T. evansi*, Lingard, 1899, India; Penning, 1899, Java. *T. brucei*, Bruce *et al.*, 1895, Zululand. *T. congolense*, Bruce *et al.*, 1913, Nyasaland; Duke, 1913, Uganda. *T. vivax*, Duke, 1913, Uganda. *T. uniforme*, Duke, 1913, Uganda. *P. bubalis*, Sheather, 1919, India. *B. buffeli*, Neveu-Lemaire, 1912, Asia.

Cabassous unicinctus = (Dasypus unicinctus).

Callithrix jacchus = Hapale jacchus *: T.*, Z. S., 1925, Brazil.

Callithrix penicillata = Hapale penicillata *: T. minasense*, Dios, Zuccarini and Werngren, 1925, Paraguay.

Callosciurus vittatus = (Sciurus vittatus).

Camel (Camelus): *T. evansi*, Evans, 1880, India. *T. evansi* var. *mbori*, Cazalbou, 1903, Senegal. *T. sudanense*, Sergent, 1904, Algeria. *T. berberum*, Sergent and Lhéritier, 1912, Algeria. *T. pecaudi* (= *T. brucei*), Balfour, 1909, Sudan; Wenyon, 1909, Sudan. *T. congolense*, Broden, 1906, Congo. *B. camelensis*, Yakimoff, Schokhor and Koselkine, 1917, Turkestan.

Canis adustus (jackal): *B. rossi*, Nuttall, 1910, Africa. *Hp. canis adusti*, Nuttall, 1910, Africa; Yakimoff, 1911, Tunis.

Canis aureus (jackal): *B. gibsoni*, Patton, 1910, India. *Hp. rotundata*, Patton, 1910, India.

Cat (Felis) *: T. brucei*, Bruce, 1895, Zululand. *T. cruzi*, Chagas, 1909, Brazil. *Hp. felis domestici*, Patton, 1908, India. *Leishmania donovani*, Sergent, Lombard and Quilichini, 1912, Algiers and ? Pittaluga, 1925, Spain.

(Catoblepas gnu) = Connochætes gnu (wildebeeste): *T. brucei*, Bruce *et al.*, 1897, Zululand. *T.* sp., Week, 1914, East Africa.

(Cavia cobaia) = Cavia porcellus (guinea-pig): *T.* sp., Kunstler, 1883 (?). *Tx. caviæ*, Carini and Migliano, 1916, Brazil.

Cavia porcellus = (Cavia cobaia).

Cephalophus grimmi (duiker): *T. brucei*, Bruce *et al.*, 1913, Nyasaland; Taute, 1913, Tanganyika. *T. vivax*, Kinghorn and Yorke, 1912, Rhodesia. *T. congolense*, Kinghorn and Yorke, 1912, Rhodesia. *T. ingens*, Bruce *et al.*, 1912, Uganda; Rodhain, Pons, Vandenbranden and Bequaert, 1912, Belgian Congo. *T. cephalophi*, Bruce *et al.*, 1913, Nyasaland. *T. theileri*, Rodhain, Pons, Vandenbranden and Bequaert, 1912, Belgian Congo. *B.* sp., Bettencourt and Borges, 1909, Africa. *P.* sp., Bruce *et al.*, 1915, Nyasaland.

Cercocebus æthiopicus (mangabey): *P. kochi*, Plimmer, 1916, Africa.

Cercocebus sp. (mangabey): *P. kochi*, Koch, 1906, Africa.

Cercopithecus albogularis : *P. kochi*, Sergent, 1908, Africa.

Cercopithecus callitrichus : *P. kochi*, Joyeux, 1913, French Guinea.

Cercopithecus cephus : *T. lewisi* var. *primatum*, Reichenow, 1917, Cameroons. *P. kochi*, Ringenbach, 1914, Africa.

Cercopithecus erythrotis : *P. kochi*, Z.S., 1925, Gold Coast.

Cercopithecus fuliginosus : *P. kochi*, Gonder and Berenberg-Gossler, 1908, Africa.

Cercopithecus mona : *P.*, Seidelin and Connal, 1914, West Africa.

Cercopithecus pygerythrus : *T. gambiense*, Bruce *et al.*, 1911, Uganda. *P. kochi*, Kinghorn and Yorke, 1912, Rhodesia.

Cercopithecus sabæus : *P. kochi*, Laveran, 1899, Africa; Martoglio, Sella and Carpano, 1910, Africa; Plimmer, 1912, West Africa.

Cercopithecus schmidti: *T.* sp., Dutton, Todd and Tobey, 1906, Belgian Congo.

Cercopithecus sp.: *T. gambiense*, Koch, 1909, Uganda. *T.* sp., Kudicke, 1906, Congo. *P. kochi*, Koch, 1906, Africa; Bruce and Nabarro, 1903, Africa; Dutton, Todd and Tobey, 1906, Africa; Ross, 1911, East Africa. *B. pitheci*, Ross, 1905, Uganda.

Cervicapra arundinum (reed buck): *T. theileri*, Kleine and Fischer, 1911, Tanganyika. *T. brucei*, Bruce *et al.*, 1903, Zululand; Bruce *et al.*, 1913, Nyasaland; Taute, 1913, East Africa. *T. congolense*, Bruce *et al.*, 1913, Nyasaland; Kleine and Fischer, 1911, East Africa. *T. ingens*, Bruce. 1909, Uganda; Bruce *et al.*, 1913, Nyasaland. *T. capræ*, Bruce, 1913, Nyasaland. *T. vivax*, Connal, 1917, West Africa; Simpson, 1918, West Africa. *T. cazalboui* (= *T. vivax*),

Rodhain, Pons, Vandenbranden and Bequaert, 1913, Belgian Congo. *T.* sp., Kleine and Fischer, 1911, Tanganyika; Weck, 1914, East Africa. *Hg.* (leucocytic), Fantham, 1921, South Africa.

Cervulus muntjac (muntjac): *B.* sp., Schein, 1923, Annam.

(Cervus aristotelis) = **Rusa rusa** (sambhar): *B.* sp., Denier, 1907, Annam.

(Cervus axis) = **Axis axis** (spotted deer): *B.* sp., Patton, 1910, India.

(Cervus dama) = **Dama dama** (fallow deer): *B. cervi* (*Theileria cervus*) Bettencourt, França and Borges, 1907, Portugal = *Theileria damœ* Bettencourt and Borges, 1909.

Cervus sika : *B. dama*, Ono and Kondo, 1923, Japan.

Cholœpus didactylus (two-toed sloth): *T.* sp., Mesnil and Brimont, 1908, Guiana, *Endotrypanum schaudinni*, Mesnil and Brimont, 1908, Guiana; Darling, 1914, Panama; Labernadie and Hubac, 1923, Guiana; Z.S., 1925, Brazil.

Chrysothrix sciureus : *T. cruzi*, Chagas, 1909, 1924, Brazil.

Citellus beecheyi = (Otospermophilus beecheyi).

Citellus eversmanni = (Spermophilus eversmanni).

Citellus guttatus = (Spermophilus guttatus).

Citellus musicus = (Spermophilus musicus).

Citellus richardsoni (ground squirrel): *T. citelli*, Watson, 1912, Canada.

Cobus defassa (water buck): *B.* sp., Rodhain, 1916, Belgian Congo.

Cobus ellipsiprymnus (water buck): *T. brucei*, Bruce *et al.*, 1913, Nyasaland; Kleine and Fischer, 1911, East Africa; Kinghorn and Yorke, 1912, Nyasaland; Stohr, 1913, Rhodesia; Taute, 1913, Tanganyika; Duke, 1913, Uganda. *T. vivax*, Kleine and Fischer, 1911, Tanganyika; Kinghorn and Yorke, 1912, Rhodesia; Duke, 1913, Uganda; Johnson, 1920, West Africa. *T. congolense*, Bruce *et al.*, 1913, Nyasaland; Kinghorn and Yorke, 1912, Rhodesia. *T. caprœ*, Bruce *et al.*, 1913, Nyasaland. *T. uniforme*, Duke, 1913, Uganda; *T. ingens*, Bruce *et al.*, 1914, Nyasaland. *T.* sp., Kleine and Fischer, 1911, Tanganyika; Weck, 1914, East Africa.

Cobus vardoni (puku): *T. vivax*, Kinghorn and Yorke, 1912, Rhodesia. *T. ingens*, Rodhain, Pons, Vandenbranden and Bequaert, 1913, Belgian Congo. *T. cazalboui* (= *T. vivax*), Rodhain, Pons, Vandenbranden and Bequaert, 1913, Belgian Congo.

Connochætes gnu = (Catoblepas gnu).

(Cricatellus phocus) = **Cricetulus migratorius** (little hamster): *T.* sp., Finklestein, 1907, Caucasus.

Cricetomys gambianus (giant rat): *B.* sp., Bruce *et al.*, 1915, Nyasaland. *Hp.* sp., Rodhain, 1915, Belgian Congo.

Cricetulus griseus (striped hamster): *T.* and *Leishmania donovani*, Young and Hertig, 1926, North China.

Cricetulus migratorius = (Cricatellus phocus).

Cricetus cricetus = (Cricetus frumentarius).

(Cricetus frumentarius) = **Cricetus cricetus** (common hamster): *T. rabinowitschi* Brumpt, 1906, France; Wittich, 1851, Germany; Koch, 1881, Germany; Chalachnikov, 1888, and Dudčenko, 1913, Russia; Rabinowitsch and Kempner, 1899, Germany; Wasielewski, 1908, Germany; Nöller, 1912, Germany. *Hp. criceti*, Nöller, 1912, Austria.

Crocidura russula (shrew): *T. crocidurœ*, Brumpt, 1923, France.

Crocuta crocuta = (Hyæna crocuta).

Cryptoprocta ferox (fossa): *Tx.* sp., Plimmer, 1915, Madagascar.

Ctenodactylus gundi (gundi): *B. quadrigemina*, Nicolle, 1907, Tunis. *Tx. gondii*, Nicolle and Manceaux, 1908, 1909, and Nicolle and Conor, 1913, Tunis.

(Cynocephalus sp.) = **Papio sp.** (baboon): *P. kochi*, Koch, 1906, Africa.

Cyon dukhunensis (Indian wild dog): *B.* sp., Plimmer, 1915, India. *Hp. canis*, Donovan (first record), India.

Dama dama =(Cervus dama).

(Dasypus novemcinctus) =Tatusia novemcincta (armadillo): *T. cruzi*, Chagas, 1912, Brazil; Torres, 1915, Brazil.

Dasypus sexcinctus (armadillo): *T. cruzi*, Torres, 1915, Brazil.

(Dasypus unicinctus) =Cabassous unicinctus (armadillo): *T. cruzi*, Torres, 1915, Brazil.

Dasyurus viverrinus (dasyure): *Hp. dasyuri*, Welsh and Barling, 1908 and 1910, Australia.

Dendromys sp. (? Insignis) : *Hp.* sp., Kleine, 1910, Tanganyika.

Dendromys sp.: *T. dendromysi*, Rodhain, 1915, Belgian Congo.

(Didelphys didelphys aurita) =Didelphys marsupialis (opossum): *Hp. didelphydis*, d'Utra and Arantes, 1916, Brazil.

Didelphys marsupialis =(Didelphys didelphys aurita).

Dog (Canis familiaris): *T. gambiense*, Gray and Tulloch, 1907, Uganda; Kopke, 1908, Isle of Principe; Koch, 1909, Uganda. *T. brucei*, Bruce *et al.*, 1915, Nyasaland. *T. pecaudi* (=*T. brucei*), Bouet, 1908, Senegal. *T. congolense*, Martin, Lebœuf and Roubaud, 1908, Congo. *T. dimorphon*, Martin, 1906, Congo. *T. cazalboui* (=*T. vivax*), Rodhain, Pons, Vandenbranden and Bequaert, 1913, Belgian Congo. *T. evansi*, Lingard, 1894, India. *T. annamense*, Blin, 1902, Tonkin. *T. venezuelense*, Rangel, 1905, Venezuela. *T. morocanum*, Delanoë, 1920, Algeria. *T. montgomeryi*, Kinghorn and Yorke, 1912, Rhodesia.

    Leishmania donovani, Nicolle and Comte, 1908, Tunis; Yakimoff, 1911, Tunis; Sergent, 1910, Algeria; Basile, 1910, Sicily; Pulvirenti, 1911, Sicily; Jemma, 1912, Sicily; Lignos, 1912, Isle of Hydra; Cardamatis, 1911, Greece; Alvarez and da Silva, 1910, Portugal; Dschunkowsky and Luhs, 1909, Transcaspia; Critien, 1911, Malta; Delanoë and Denis, 1919, Morocco; Martinez, 1914, Spain; Lafont and Heckenroth, 1915, Senegal; Yakimoff and Schokhor, 1915, Turkestan; Castellani, 1913, Ceylon (?). *Leishmania tropica*, Neligan, 1913, Teheran; Yakimoff and Schokhor, 1914, Turkestan; Gachet, 1915, Turkestan.

    B. canis, Piana and Galli-Valerio, 1895, Europe. *B. gibsoni*, Patton, 1910, India. *B. vitalli*, Pestana, 1910, Brazil. *Tx. canis*, Mello, 1910, Europe; Carini, 1911, Brazil; Yakimoff, 1911, Germany; Carini and Maciel, 1913, Brazil; Blanc, 1917, Tunis; Federovitch, 1916, Black Sea; Boez, 1921, Strasburg; Donovan (first record), India.

    Hp. canis, Bentley and James, 1905, India; Gerrard and Wenyon, 1906, Malaya; Christophers, 1907, India; Wenyon, 1911, Bagdad; Lebœuf and Roubaud, 1910, West Africa; Yakimoff, 1911, Tunis; Yakimoff and Schokhor, 1917, Turkestan; Leger, 1912, Corsica; Basile, 1911, Italy; Sergent and Senevet, 1912, Algeria; Mathis and Leger, 1909, Tonkin; Martoglio, 1913, Eritrea; Pringault, 1920, France. *Hp. rotundata* var. *familiaris*, Martoglio, 1913, Eritrea. *P. canis*, Castellani and Chalmers, 1908, Ceylon.

Donkey (Equus asinus ?): *T. evansi*, Evans, 1880, India. *T. sudanense*, Roger and Greffulhe, 1905, North Africa. *T. venezuelense*, Tejera, 1920, Venezuela. *T. brucei*, Bruce *et al.* 1895, Zululand. *T. togolense*, Schilling, 1901, Togoland. *T. pecaudi* (=*T. brucei*), Cazalbou, 1910, Senegal. *T. congolense*, Broden, 1904, Congo. *T. dimorphon* (=*T. congolense* ?), Martin, 1906, Senegal. *T. vivax*, Hornby, 1919, Rhodesia. *T. cazalboui* (=*T. vivax*), Bouffard, 1907, Senegal. *T. equiperdum*, Schneider and Bouffard, 1899, Algeria. *T. equinum*, Vital, 1907, Brazil. *B. asini*, Dschunkowsky and Luhs, 1909, Transcaucasia.

Edible rat (Steatomys edulis ?): *B.* sp., Bruce *et al.*, 1915, Nyasaland.

Elephant (Elephas sp.): *T. evansi*, Evans, 1880, India; Lingard, 1894, India. *T. elephantis* (=*T. brucei* ?), Bruce *et al.*, 1909, Uganda.

Eliomys quercinus =Myoxus nitela.

Epomophorus franqueti : *P.* sp., Rodhain, 1915, Belgian Congo.

Epomophorus gambianus : *P. pteropi*, Leger, A. and M., 1914, Senegal.

Eptesicus capensis = (Vespertilio capensis).

Eptesicus serotinus = (Vespertilio serotinus).

Equus burchelli granti (Burchell's zebra): *B.* sp., Ross, P. H., 1907, Uganda; Ross, P. H., 1911, East Africa; Kudicke, quoted by Ollweg and Manteufel, 1912, Africa.

(Erinaceus algirus) =Æthechinus algirus (hedgehog): *B. ninense*, Yakimoff, 1910, Russia; *B. weissi*, Galli-Valerio, 1911, Tunis.

Erinaceus europæus (hedgehog): *B. ninense*, Yakimoff, 1910, Russia.

Evotomys saturatus : *T: evotomys*, Hadwen, 1912, Canada.

Felis leo (lion).

(Fennecus dorsalis) =Vulpes dorsalis (fox ?): *B. bauryi* =*Nuttallia bauryi*, Leger and Bédier, 1922, French Sudan.

Funambulus palmarum =(Sciurus palmarum) (palm squirrel): *T. indicum*, Lühe, 1906, India.

Funambulus pennantii (? squirrel): *Hp. funambuli*, Patton, 1906, India.

(Galago demidoffi) =Hemigalago demidoffi (lemur): *T.*, Martin, Lebœuf and Roubaud, 1909, Congo.

Gazella grantii : *B. stordii*, França, 1912, Abyssinia.

Gazella sp. : *B. stordii*, Carpano, 1913, Eritrea.

Gazella thomsoni : *B.* sp., Ross, P. H., 1911, East Africa.

(Gerbillus indicus) =Tatera indica (Indian gerbil): *Hp. gerbilli*, Christophers, 1905, India.

Giraffe (Giraffa) : *Hg.* (leucocytic), Fantham, 1919, South Africa.

Goat (Capra hircus ?) : *T. brucei*, Bruce et al., 1915, Nyasaland. *T. pecaudi* (=*T. brucei*), Pecaud, 1909, Senegal. *T. congolense*, Martin, Lebœuf and Roubaud, 1909, Congo. *T. dimorphon* (=*T. congolense* ?), Martin, 1906, Congo. *T. capræ*, Kleine, 1910, East Africa; Fehlandt, 1911, East Africa; Kleine and Taute, 1911, East Africa; Bruce et al., 1914, Nyasaland. *T. vivax*, Ziemann, 1905, Cameroons; *T. cazalboui* (=*T. vivax*), Bouet, 1908, Senegal. *T. gambiense*, Kleine and Eckard, 1913, Central Africa. *B. hirci*, Dschunkowsky and Luhs, 1909, Transcaucasia. *Theileria hirci*, Dschunkowsky and Urodschevich, 1924, Serbia, and Lestoquard, 1924, Algeria. *Leishmania caprae* (?), Curson, 1926, S. Africa (verbal communication).

Golunda campanæ (jerboa rat): *B. campanæ*, Leger and Bédier, 1923, Senegal.

Gorilla gorilla =(Anthropopithecus gorilla).

Hapale jacchus (marmoset): *T. minasense*, Chagas, 1908, 1909, Brazil.

Hapale penicillata (marmoset): *T. minasense*, Chagas, 1909, Brazil; Cerquéira, 1924, Brazil.

Hare (Lepus) : *B. leporis*, Dschunkowsky and Luhs, 1909, Transcaucasia.

Hemigalago demidoffi =(Galago demidoffi).

Herpestes calera : *B. legeri*, Bédier, 1924, Africa.

Herpestes edwardsi =(Herpestes mungo).

Herpestes ichneumon (Egyptian mongoose): *B. herpestidis*, França, 1908, Portugal.

(Herpestes mungo) =Herpestes edwardsi (Indian mongoose): *B.* sp., Patton, 1909, India.

Hippopotamus : *T.* sp., Kleine and Taute, 1911, East Africa.

Hipposiderus speoris : *T.*, Donovan (first record), India.

Hipposiderus tridens (bat): *T. morinorum*, Leger and Baury, 1923, Senegal.

Hippotragus equinus (roan antelope): *T vivax*, Duke, 1923, Uganda. *T. congolense*, Kinghorn and Yorke, 1912, Rhodesia; Davey, 1916, Tanganyika. *T. theileri*, Rodhain, Pons, Vandenbranden and Bequaert, 1913, Belgian Congo. *T. cazalboui* (=*T. vivax*), Rodhain, Pons, Vandenbranden and Bequaert, 1913, Belgian Congo. *B. hippotragi*, Todd and Wolbach, 1912, Gambia.

Hippotragus niger (sable antelope): *T.* sp., Weck, 1914, East Africa.

Horse (Equus caballus) : *T. evansi*, Evans, 1880, India. *T. annamense*, Blanchard, 1888, Tonkin. *T. sudanense*, Chauvrat, 1892, Algeria. *T. evansi* var. *mbori*, Cazalbou, 1903, Senegal. *T. equinum*, Voges, 1901, South America. *T. hippicum*, Darling, 1910, Panama. *T. venezuelense*, Rangel, 1905, Venezuela.

T. marocanum, Sergent, Lhéritier and Belleval, 1915, Morocco. *T. berberum*, Sergent and Lhéritier, 1912, North Africa. *T. brucei*, Bruce *et al.*, 1895, Zululand. *T. togolense*, Schilling, 1901, Togoland. *T. vivax*, Yorke and Blacklock, 1911, Rhodesia. *T. congolense*, Bruce *et al.*, 1914, Nyasaland. *T. pecaudi* (= *T. brucei*), Cazalbou, 1900, Senegal. *T. cazalboui* (= *T. vivax*), Bouet, 1907, Senegal. *T. dimorphon* (= *T. congolense* ?), Dutton and Todd, 1903, Gambia. *T. equiperdum*, Rouget, 1896, North Africa. *B. caballi*, Nuttall, 1910, Europe. *B. equi*, Laveran, 1901, Europe. *P. equi*, Castellani and Chalmers, 1913, Ceylon. *Leishmania* (?), Richardson, 1926, Uganda.

(Hyæna crocuta) =Crocuta crocuta : *Hp. chattoni*, Leger, 1912, Senegal. *Hp. canis*, Leger, 1912, Senegal.

Hydrochœrus capybara (capybara): *T. venezuelense*, Tejera, 1920, Venezuela. *T. equinum*, Lutz 1907, Brazil, and Migone, 1910, Paraguay.

Hyena : *T. brucei*, Bruce *et al.*, 1895, Zululand. *T. congolense*, Bruce *et al.*, 1913, Nyasaland.

Ictonyx zorilla : *P. roubaudi*, Leger and Bédier, 1923, Senegal.

Jaculus gordoni = Jaculus jaculus.

Jaculus jaculus = Jaculus gordoni (jerboa): *Hp. jaculi* =*Hp. balfouri*, Balfour, 1905, Sudan.

Jaculus (Johnstoni ?) : *Hp.* sp., Rodhain, Pons, Vandenbranden and Bequaert, 1913, Belgian Congo.

Jaculus orientalis (large Egyptian jerboa): *Hp. balfouri*, Laveran, 1905, Tunis; Z.S., 1925, Egypt.

Lavia frons = (Megaderma frons).

Lemniscomys barbarus pulchellus = (Arvicanthis barbarus pulchellus).

Lemniscomys zebra = (Arvicanthis zebra).

(Lepus cuniculus) =Oryctolagus cuniculus (rabbit): *Hp. cuniculi*, Sangiorgi, 1914, Italy.

Lepus nigricollis (black-naped hare): *Hp. leporis*, Patton, 1908, India.

(Lepus sylvaticus) = Sylvilagus sylvaticus (American " rabbit "): *T. leporis sylvatici*, Watson, 1912, Canada.

Leuconœ daubentonii = (Vespertilio daubentonii).

Limnotragus spekei = (Tragelaphus spekei).

Lion (Felis leo) : *T.* sp., Weck, 1914, East Africa. *B.* sp., Leger and Bédier, 1922, French Sudan.

Macaca fascicularis = (Macacus cynomolgus).

(Macacus cynomolgus) =Macaca fascicularis (common macaque): *P. cinomolgi*, Mayer, 1907, Asia. *P. inui*, Halberstaedter and Prowazek, 1907, Borneo. *Hg. blanchardi*, Langeron, 1920, Asia.

Macacus lasiotis tcheliensis : *P. inui*, Mathis and Leger, 1911, Tonkin.

Macacus mulatta = (Macacus rhesus).

Macacus nemestrinus : *P. inui*, Halberstaedter and Prowazek, 1907, Borneo.

Macacus pileatus : *B. cellii*, Castellani and Chalmers, 1910, Ceylon.

(Macacus rhesus) =Macacus mulatta : *T. rhesii*, Terry, 1911, America. *P. inui*, Mathis and Leger, 1911, Tonkin; Bruce and Nabarro, 1903, Africa; Chimisso, 1922, Italy.

Macacus sinicus : *P. inui*, Donovan, 1920, India.

Marmot (Marmota sp.): *Hp. plicata marmotœ*, Martoglio, 1913, Eritrea.

Marmota marmota = (Arctomys marmota).

(Megaderma frons) =Lavia frons (African bat): *T. megadermœ*, Wenyon, 1909, Sudan.

Meles meles = (Meles taxus).

(Meles taxus) =Meles meles (badger): *T. pestani*, Bettencourt and França, 1905, Portugal.

Microtus agrestis (field vole): *Hp. microti*, Coles, 1914, England.

(Microtus amphibius) =Arvicola amphibius (water vole): *B. microti*, Coles, 1914, England.

(Microtus arvalis) = Arvicola arvalis (field vole): *T. microti*, Laveran and Pettit, 1909, France; Yakimoff and Korssak, 1910, Russia (?); Lavier, 1921, France. *Hp. arvalis*, Lavier, 1921, France.

Microtus incertus (field vole): *B. microti*, França, 1909, Portugal.

Midas midas (yellow-banded marmoset): *T. clevei*, Leger and Porry, 1918, French Guiana.

Miniopterus schreibersi (bat): *T. vespertilionis*, Battaglia, 1904, Italy; Dionisi, 1899, Italy. *P. melanipherum*, Dionisi, 1898, Italy; Schingareff, 1906, Russia.

Monkey : *P. kochi*, Bouillez, 1916, Central Africa.

Mouse (Mus musculus ?) : *B.* sp., Bruce *et al.*, 1911, Uganda. *Hp. musculi*, Porter, 1908, England; Sangiorgi, 1912, Italy; Yakimoff and Schokhor, 1917, Russia, *Tx. musculi*, Sangiorgi, 1913, Italy.

Mule : *T. evansi*, Evans, 1880, India. *T. annamense*, Blanchard, 1888, Tonkin. *T. hippicum*, Darling, 1910, Panama. *T. venezuelense*, Tejera, 1920, Venezuela. *T. brucei*, Bruce *et al.*, 1895, Zululand. *T. pecaudi* (= *T. brucei*), Bouffard, 1908, Senegal. *T. congolense*, Hornby, 1919, Rhodesia. *T. dimorphon*, (= *T. congolense* ?), Martin, 1906, Congo. *T. vivax*, Hornby, 1919, Rhodesia. *B. rossica*, Yakimoff, Schokhor and Koselkine, 1917, Turkestan.

(Mus agrarius) =Apodemus agrarius (striped field mouse): *T. korssaki*, Yakimoff, Kohl-Yakimoff and Korssak, 1910, Siberia. *B.* sp., Yakimoff, Kohl-Yakimoff and Korssak, 1910, Siberia.

(Mus alexandrinus) =Rattus rattus alexandrinus : *T. lewisi*, Yakimoff, 1911, Tunis. *Hp. muris*, Kusama, Kasai and Kobayashi, 1919, Japan; Cleland, 1906, Australia.

(Mus coucha) =Rattus coucha : *T. eburneense*, Delanoë, 1915, West Africa.

(Mus cunninghami) =Pseudomys cunninghami : *Hp.* sp., Kleine, 1910, Tanganyika.

(Mus decumanus =Mus norvegicus) =Rattus norvegicus (brown rat): *T. lewisi*, Lewis, 1877, India; Yakimoff, 1911, Tunis; Petrie and Avari, 1909, Bombay. *B. decumani*, Macfie, 1915, West Africa. *Hp. muris*, Balfour, 1906, Sudan; Carini, 1910, Brazil; Yakimoff, 1911, Tunis; Coles, 1914, England; Johnston, 1909, Australia; Adie, 1906, India; Kusama, Kasai and Kobayashi, 1919, Japan; Darling, 1912, Panama; Wenyon, 1911, Bagdad.

(Mus macleari) =Rattus macleari : *T. lewisi*, Durham, 1908, Christmas Isle.

Mus manei : *T.*, Donovan (first record), India.

(Mus maurus) =Rattus maurus : *T. lewisi*, Martin, Lebœuf and Roubaud, 1909, West Africa.

(Mus morio) = Rattus morio : *T. duttoni*, Thiroux, 1905, Senegal.

Mus musculus (mouse): *T. musculi*, 1906, Panama; Pricolo, 1906, Italy. *Tx. musculi*, Sangiorgi, 1913, Italy.

(Mus niveiventer) =Rattus niveiventer : *T. lewisi*, Lingard, 1906, India.

(Mus norvegicus =Mus decumanus) =Rattus norvegicus.

(Mus rattus) =Rattus rattus (black rat): *T. lewisi*, Lewis, 1877, India; Chaussat, 1850, France; Durham, 1908, Christmas Isle; Petrie and Avari, 1909, Bombay. *Hp. muris*, Kusama, Kasai and Kobayashi, 1919, Japan; Stammers, 1920, England. *Tx. ratti*, Sangiorgi, 1915, Italy.

(Mus rufescens) =Rattus rufescens : *T. lewisi*, Lewis, 1877, India. *Hp. muris*, Donovan (first record), India.

(Mus sylvaticus) =Apodemus sylvaticus : *T. grosi*, Laveran and Pettit, 1909, France; Gros, 1845, Russia (?). *B.* sp., Coles, 1914, England. *Hp. sylvatici*, Coles, 1914, England.

Muscardinus avellanarius = (Myoxus avellanarius).

(Mustela putorius) =Putorius putorius (weasel): *Microsoma mustelœ*, Lebedeff and Tscharnotzky, 1911, Russia.

(Mycetes seniculus) =Alouatta senicula : *Tx.* sp., Thézé, 1916, French Guiana.

(Mycetes ursinus) =Alouatta ursina (howler monkey): *T. venezuelense*, Tejera, 1920, Venezuela.

Myoprocta acouchy (agouti): *T. acouchii*, Brimont, 1909, Guiana.

Myotis capaccinii (capaccini bat): *P. melaniferum*, Dionisi, 1898, Italy.

Myotis muricola = (Vespertilio muricola).

(Myotis murinus) = Myotis myotis (long-eared bat): *T. nicolleorum*, Sergent, 1905, North Africa.

Myotis myotis = (Myotis murinus = Vespertilio murinus): *P. murinum*, Dionisi, 1898, Italy.

Myotis nattereri = (Vesperugo nattereri).

(Myoxus avellanarius) = Muscardinus avellanarius (common dormouse): *T. myoxi*, Blanchard, 1903, Europe; Galli-Valerio, 1903, Switzerland. *B. myoxi*, Franchini, 1924, Italy.

Myoxus murinus : *P. rigoleti*, Leger and Bédier, 1922, Senegal.

Myoxus nitela = Eliomys quercinus (garden dormouse): *T. blanchardi*, Brumpt, 1905, France; França, 1909, Portugal; Biot, 1909, France; Laveran and Pettit, 1910, France. *Herpetomonas myoxi*, Laveran and Franchini, 1921, Italy.

Nesokia bandicota : *T.*, Donovan (first record), India.

Nesokia gigantea : *T. bandicotti*, Lingard, 1904, India.

Nyctalus noctula = (Vesperugo noctula).

Nycteris hispida : *T. heybergi*, Rodhain, 1923, Belgian Congo.

Odocoileus chiriquensis (white-tailed deer): *B. bigemina*, Clark, 1918, and Clark and Zetek, 1925, Panama.

(Oribia scoparia) = Ourebia oribi (oribi): *T. brucei*, Bruce *et al.*, 1913, Nyasaland. *T. congolense*, Bruce *et al.*, 1913, Nyasaland. *T. capræ*, Bruce *et al.*, 1913, Nyasaland. *T. ingens*, Bruce *et al.*, 1913, Nyasaland.

Oryctolagus cuniculus = (Lepus cuniculus).

Oryx beisa (beisa): *B.* sp., Ross, P. H., 1911, East Africa.

Oryx callotis : *B.* sp., Ross, P. H., 1911, East Africa.

(Otospermophilus beecheyi) = Citellus beecheyi (ground squirrel): *T. otospermophili*, Wellman and Wherry, 1910, California. *Hp. citellicola*, Wellman and Wherry, 1910, California.

Otter (Lutra sp. ?): *T.* sp., Fehlandt, 1911, Tanganyika.

Ouakaria calva = (Brachyurus calva).

Ourebia oribi = (Oribia scoparia).

Ox (Bos taurus ?): *T. evansi*, Lingard, 1899, India. *T. annamense*, Schein, 1907, Tonkin. *T. gambiense*, Bruce *et al.*, 1911, Uganda; Blacklock and Yorke, 1915, West Africa; Kleine and Eckard, 1913, Central Africa. *T. brucei*, Bruce *et al.*, 1895, Zululand. *T. pecaudi* (= *T. brucei*), Cazalbou, 1910, Senegal. *T. togolense*, Schilling, 1901, Togoland. *T. congolense*, Broden, 1906, Congo. *T. dimorphon* (= *T. congolense* ?), Martin, 1906, Congo. *T. vivax*, Ziemann, 1905, Cameroons. *T. cazalboui* (= *T. vivax*), Cazalbou, 1904, Senegal. *T. uniforme*, Bruce *et al.*, 1911, Uganda. *T. ingens*, Bruce *et al.*, 1909, Uganda. *T. montgomeryi*, Montgomery and Kinghorn, 1909, Rhodesia. *T. theileri*, Theiler, 1902, South Africa. *P. bubalis*, Sheather, 1919, and Edwards, 1925, India. *B. bigemina*, Smith and Kilborne, 1893, Texas. *B. bovis*, Babes, 1888, Europe. *B. divergens*, M'Fadyean and Stockman, 1911, England, *B. argentina*, Linières, 1898, South America. *B. mutans*, Theiler, 1907, South Africa. *Theileria parva*, Theiler, 1904, South Africa. *Hg. bovis*, Martoglio and Carpano, 1906, Eritrea. *Hg. boum*, Legras, 1918, Algeria.

Papio sp. = (Cynocephalus sp.).

Papio sphinx (baboon): *P.*, Seidelin and Connal, 1914, West Africa. *Hæmogregarina cynomolgi* var. *papio*, Leger and Bédier, 1922, French Sudan.

Perameles nasuta (Australian bandicoot): *Hp. peramelis*, Welsh and Dalyell, 1910, Australia.

Perodicticus sp. (potto): *T. lewisi* var. *primatum*, Reichenow, 1917, Cameroons.

Peromyscus maniculatus (American field mouse): *T. peromysci*, Watson, 1912, Canada.

Peromyscus nebrascensis (American field mouse): *T. peromysci*, Watson, 1912, Canada.

Petaurus scieureus (flying opossum): *Hp. petauri*, Welsh, Dalyell and Burfitt, 1908 and 1910, Australia.

Petrodromus tetradactylus (elephant shrew): *P. brodeni*, Rodhain, Pons, Vandenbranden and Bequaert, 1913, Belgian Congo. *Hp.* sp., Rodhain, Pons, Vandenbranden and Bequaert, 1913, Belgian Congo.

Petrodromus venustus (elephant shrew): *T. petrodromi*, Bruce *et al.*, 1915, Nyasaland. *P.* sp., Bruce *et al.*, 1915, Nyasaland.

Phacocœrus æthiopicus (wart hog): *T. brucei*, Bruce *et al.*, 1913, Nyasaland; Kinghorn and Yorke, 1912, Rhodesia. *T. congolense*, Bruce *et al.*, 1913, Nyasaland; Simpson, 1918, West Africa. *T. simiæ*, Bruce *et al.*, 1913, Nyasaland.

Phyllostoma perspicillatum =Arlibeus perspicillatus (South American bat): *T. phyllostomæ*, Cartaya, 1910, South America.

Pig (Sus scrofa ?) : *T. brucei*, Macfie, 1916, Nigeria. *T. pecaudi* (= *T. brucei*), Bouet, 1908, Senegal. *T. congolense*, Bruce *et al.*, 1914, Nyasaland. *T. dimorphon* (= *T. congolense* ?), Martin, 1906, Congo. *B. trautmanni*, Knuth and du Toit, 1918, East Africa. *B. ovis*, Sparapani, 1917, Italy.

Pipistrellus pipistrellus = (Vesperugo pipistrellus).

Pithecus entellus = (Semnopithecus entellus).

Pitymys savii (burrowing vole): *Hp. pitymidis*, Splendore, 1918, Italy.

Plecotus auritus (long-eared bat): *T. vespertilionis*, Bettencourt and França, 1905, Portugal.

Presbytes pileatus : *P. semnopitheci*, Z.S., 1925, Assam.

Procyon lotor (racoon): *B.* sp., Wenyon and Scott, 1926, North America (Z.S.).

Pseudomys cunninghami = (Mus cunninghami).

Pteromys petaurista (flying squirrel): *Hp.*, Donovan (first record), India.

Pteropus edwardsii (flying fox): *P. pteropi*, Mackie, 1913, India.

Pteropus gouldi (flying fox): *P. pteropi*, Breinl, 1912, West Australia.

Pteropus hypomelanus : *P. pteropi*, Z.S., 1925, Java.

Pteropus medius (flying fox): *T.* sp., Donovan (quoted by Laveran and Mesnil, 1912), India.

Pteropus natalis (flying fox): *P.* sp., Durham, 1908, Christmas Island.

Putorius putorius = (Mustela putorius).

Rabbit (Oryctolagus cuniculus ?) : *T. nabiasi*, Railliet, 1895, France; Jolyet and Nabias, 1891, France; Petrie, 1904, England; Bosc, 1904, France; Laveran and Mesnil, 1904, Spain; Bettencourt and França, 1906, Portugal; Manca, 1906, Sardinia; Ashworth and MacGowan, 1909, Scotland; Jouan, 1911, France. *Hp. cuniculi*, Sangiorgi, 1914, Italy; Bourret, 1911, Senegal; Porter, 1918, and Fantham, 1919, South Africa. *Tx. cuniculi*, Splendore, 1908, Brazil; Bourret, 1911, Senegal.

Rangifer tarandus (reindeer): *B.* sp., Kerzelli, 1909, Russia.

Raphiceros campestris (stein buck): *T. brucei*, Bruce *et al.*, 1903, Zululand.

Rat, white =Rattus norvegicus : *B. muris*, Fantham, 1905, England. *Hp. perniciosum*, Miller, 1908, America. *Tx. ratti*, Sangiorgi, 1915, Italy.

Rattus alexandrinus = (Mus alexandrinus).

Rattus coucha = (Mus coucha).

Rattus macleari = (Mus macleari).

Rattus maurus = (Mus maurus).

Rattus morio = (Mus morio).

Rattus niveiventer = (Mus niveiventer).

Rattus norvegicus = (Mus norvegicus =Mus decumanus).

Rattus rattus = (Mus rattus).

Rattus rufescens = (Mus rufescens).

Ratufa indica (giant squirrel): *P. ratufæ*, Donovan, 1920, India.

Rhabdomys pumilio = (Arvicanthis pumilio).

Rusa rusa = (Cervus aristotelis).

Sciurus griseimanus : *P. vassali*, Laveran, 1905; Vassal, 1905, 1907, Annam.

(Sciurus palmarum) = Funambulus palmarum (palm squirrel): *T. indicum*, Lühe, 1906, India; Donovan (quoted by Laveran and Mesnil), Madras.

Sciurus sp. (squirrel): *P. vassali*, Vassal, 1907, Annam.

(Sciurus vittatus) = Callosciurus vittatus (striped squirrel): *P. vassali*, Vassal, 1907, Annam.

Sciurus vulgaris (common squirrel): *Tx. sciuri*, Coles, 1914, England.

Scotophilus kuhli = (Vespertilio kuhlî).

(Semnopithecus entellus) = Pithecus entellus (entellus monkey or hanuman): *P. semnopitheci*, Knowles, 1919, India.

Serval (Felis serval) : *T.* sp., Week, 1914, East Africa.

Sheep (Ovis aries ?): *T. pecaudi* (= *T. brucei*), Pecaud, 1909; Cazalbou, 1910, Senegal. *T. congolense*, Broden, 1904, Congo. *T. dimorphon* (= *T. congolense* ?), Martin, 1906, Congo. *T. vivax*, Ziemann, 1905, Cameroons. *T. cazalboui* (= *T. vivax*), Bouet, 1908, West Africa. *T. capræ*, Fehlandt, 1911, East Africa. *T. gambiense*, Kleine and Eckard, 1913, Central Africa. *T. melophagium*, Woodcock, 1910, England; Behn, 1911, Germany; Nöller, 1917, Germany; Hoare, 1921, England; Douwes, 1920, Holland. *B. ovis*, Starcovici, 1893, Europe; Babes, 1888, Europe. *B.* sp., Rodhain, 1916, Africa. *Theileria* sp., Mason, 1915, 1916, Egypt. *B.* spp., and *Theileria hirci*, Lestoquard, 1924, 1925, Algeria.

Simia satyrus (orang-outang): *P. pitheci*, Halberstaedter and Prowazek, 1907, Borneo; Laveran, 1905, Asia; Shibayawa, 1910; Dodd, 1913; Donovan (first record), Indian menagerie; Z.S., 1925, Borneo and Sumatra.

Sorex vagrans (shrew): *T. soricis*, Hadwen, 1912, Canada.

(Spermophilus eversmanni) – Citellus eversmanni : *T. spermophili*, Laveran, 1911; Grüner, 1910, Russia.

(Spermophilus guttatus) – Citellus guttatus : *T. spermophili*, Laveran, 1911; Chalachnikow, 1888, Russia.

(Spermophilus musicus) – Citellus musicus : *T. spermophili*, Laveran, 1911; Chalachnikow, 1888, Russia.

Steatomys pratensis (fat mouse): *T.* sp., Plimmer, 1912, South Africa.

(Strepsiceros capensis) – Strepsiceros strepsiceros (koodoo): *T. brucei*, Bruce *et al.*, 1895, Zululand. *T. congolense*, Bruce *et al.*, 1913, Nyasaland, Kinghorn and Yorke, 1912, Rhodesia. *T. capræ*, Bruce *et al.*, 1914, Nyasaland. *T. cazalboui* (– *T. vivax*), Rodhain, Pons, Vandenbranden and Bequaert, 1913, Belgian Congo.

Strepsiceros strepsiceros = (Strepsiceros capensis).

Sylvilagus sylvaticus = (Lepus sylvaticus).

Tachyglossus aculeatus (echidna): *Theileria tachyglossi*, Priestley, 1915, Australia.

Talpa cæca (mole): *T. talpæ*, França, 1911, Portugal.

Talpa europæa (mole): *T. talpæ*, Nabarro; Petrie, 1905, England; Thomson, 1906, England; França, 1911, Portugal. *B. talpæ*, Galli-Valerio, 1913, Italy. *Elleipsisoma thomsoni*, França, 1910, Portugal; Thomson, J. D., 1906, England.

Talpa sp. (mole): *Tx. talpæ*, Prowazek, 1910, Japan.

Tamandua tridactyla (tamandua): *T. legeri*, Mesnil and Brimont, 1910, French Guiana.

Tatera indica = (Gerbillus indicus).

Tatera lobengula (gerbil): *T. lewisi*, Fantham, 1926, South Africa.

Tatusia novemcincta = (Dasypus novemcinctus).

Taurotragus oryx (eland): *T. brucei*, Taute, 1913, Tanganyika; Davey, 1916, Tanganyika. *T. capræ*, Bruce *et al.*, 1913, Nyasaland. *T. congolense*, Bruce *et al.*, 1913, Nyasaland; Kinghorn and Yorke, 1912, Rhodesia; Davey, 1916, Tanganyika. *T.* sp., Week., 1914, East Africa. *Theileria* sp., Lichtenheld, 1910, Africa.

Tragelaphus scriptus (bush buck): *B.* sp., Ross, P. H., 1911, East Africa. *T. brucei*, Bruce *et al.*, 1897, Zululand; Kleine and Fischer, 1911, East Africa; Kinghorn and Yorke, 1912, Rhodesia; Taute, 1913, Tanganyika. *T. capræ*, Bruce *et al.*, 1913, Nyasaland. *T. vivax*, Bruce *et al.*, 1911, Uganda; Kleine and Fischer, 1911, East Africa. *T. cazalboui* (= *T. vivax*), Rodhain, Pons, Vandenbranden and Bequaert, 1913, Belgian Congo. *T. uniforme*, Duke, 1912, Uganda; Fraser and Duke, 1912, Uganda. *T. multiforme*, Kinghorn and Yorke, 1912, Rhodesia. *T. congolense*, Kinghorn and Yorke, 1912, Rhodesia; Bruce *et al.*, 1913; Nyasaland; Rodhain, Pons, Vandenbranden and Bequaert, 1913, Belgian Congo; Kleine and Eckard, 1913, Uganda. *T. dimorphon* (= *T. congolense* ?), Dutton, Todd and Kinghorn, 1907, Gambia; Montgomery and Kinghorn, 1908; Rhodesia; Johnson, 1920, West Africa. *T. theileri* (= *T. tragelaphi* ?), Dutton, Todd and Tobey, 1906, Gambia. *T. ingens*, Bruce *et al.*, 1909, Uganda; Fraser and Duke, 1912; Uganda; Rodhain, Pons, Vandenbranden and Bequaert, 1913, Belgian Congo. *T.* sp., Kleine and Fischer, 1911, Tanganyika; Weck, 1914, East Africa.

(Tragelaphus spekei) =**Limnotragus spekei** (Speke's antelope): *T. gambiense*, Duke, 1912, Uganda. *T. brucei*, Duke, 1921, Uganda. *T. vivax*, Duke, 1912, Uganda. *T. uniforme*, Duke, 1912 and 1923, Uganda. *T. tragelaphi*, Duke, 1912, Uganda; Kinghorn and Yorke, 1912, Rhodesia. *T. ingens*, Duke, 1912, Uganda.

Tragelaphus sylvaticus (bush buck): *T.* sp., Dutton, Todd and Tobey, 1906, Gambia.

Tragulus javanicus (chevrotain): *T.* sp., Dodd, 1912, Zoological Gardens, Sydney.

Vampyrops lineatus (striped bat): *T.* sp., Iturbe and Gonzalez, 1916, Venezuela.

(Vespertilio capensis) =**Eptesicus capensis**: *P. murinum*, Bowhill, 1906, South Africa.

(Vespertilio daubentonii) =**Leuconœ daubentonii**: *P. murinum*, Schingareff, 1906, Russia.

(Vespertilio kuhli) = **Scotophilus kuhli**: *T. vespertilionis*, Sergent, 1905, North Africa; Yakimoff, 1911, Tunis; Gonder, 1910, Italy. *B. vesperuginis*, Dionisi, 1898, Italy; Gonder, 1906, Africa.

(Vespertilio muricola) =**Myotis muricola**: *P. mackiei*, Melo and Sa, 1916, Portuguese India.

(Vespertilio murinus) =**Myotis myotis**: *P. murinum*, Dionisi, 1899, Italy.

(Vesperugo abramus) =**Pipistrellus abramus**: *P. monosoma*, Vassal, 1907, Annam.

(Vesperugo nattereri) = **Myotis nattereri**: *T. vespertilionis*, Bettencourt and França, 1905, Portugal; Gonder, 1910, Italy.

(Vesperugo noctula) =**Nyctalus noctula**: *T. vespertilionis*, Battaglia, 1906, Italy; Gonder, 1910, Italy. *B. vesperuginis*, Dionisi, 1898, Italy; Neumann, 1908, Europe; Berestneff, 1903, Europe; Galli-Valerio, 1904, Europe.

(Vesperugo pipistrellus) =**Pipistrellus pipistrellus**: *T. vespertilionis*, Petrie, 1905, England; Kisskalt, 1905, Europe; Bettencourt and França, 1905, Portugal; Mettam, 1907, Ireland; Gonder, 1910, Italy; Coles, 1914, England; Franchini, 1921, Italy. *Schizotrypanum pipistrelli*, Chatton and Courier, 1921, Alsace. *B. vesperuginis*, Dionisi, 1898, Europe; Kisskalt, 1905, Europe; Coles, 1914, England.

(Vesperugo serotinus) =**Eptesicus serotinus**: *T. vespertilionis*, Bettencourt and França, 1905, Portugal.

Viverra civetta (civet): *B. civettæ*, Leger, A. and M., 1920, Senegal.

Vulpes dorsalis = **(Fennecus dorsalis).**

Xerus erythropus (Ethiopian ground squirrel): *T. xeri*, Leger and Baury, 1922, Senegal.

Yak (Polphagus grunniens): *B.* sp., Yakimoff, Kohl-Yakimoff and Korssak, 1910, Siberia.

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Acanthogenys rufigularis (honey-sucker): *H.*, Cleland, 1915, Australia.

Accentor collaris (accentor): *H.*, Galli-Valerio, 1902, Europe.

Accipiter nisus =**(Falco nisus)** (sparrow hawk): *T.*, Mezincescu, 1909, Roumania.

H. danilewskyi var. *tinnunculus*, Galli-Valerio, 1919, Europe. *H.*, Ziemann, 1898, Europe. *L.*, Mezincescu, 1909, Roumania. *L. mathisi*, França, 1912, Portugal; Franchini, 1924, Italy.

Acomus erythrophthalmus (crestless fireback pheasant): *P. præcox*, Z.S., 1925, Malacca.

Acridotheres tristis (myna): *T.*, *H.*, and *L.*, Maya and David, 1912, Mauritius.

Ædon megarhyncha = (Erithacus luscinia).

Agapornis pullaria (red-headed lovebird): *T.*, Z.S., 1925, West Africa.

Agelæus icterocephalus (hang-nest): *H.*, Plimmer, 1913, Mexico.

Agelæus phœniceus (hang-nest): *T. confusum*, Lühe, 1906. *T. avium*, Novy, McNeal, 1905, North America. *H.*, Galli-Valerio, 1902, Europe. *P.*, Opie, 1898, North America.

Agelæus thilius = (Cacicus chrysopterus).

Aidemosyne malabarica (white-throated munia): *P. præcox*, Z.S., 1925, India.

Alopochen ægyptiacus = (Chenalopex ægyptiaca) .

Alario alario (finch): *L.*, Plimmer, 1916, and Z.S., 1925, South Africa; *H.*, Z.S., 1925, South Africa.

(Alauda arborea) = Lullula arborea (wood lark): *H.*, Galli-Valerio, 1902, Europe.

Alauda arvensis (skylark): *H. alaudæ*, Celli and San Felici, 1891, Italy; Schaudinn, quoted by Prowazek, 1911, Germany; Ziemann, 1898, Italy; Wasielewski, 1896, Germany; Labbé, 1894, France; Grassi and Feletti, 1890, Sicily; Laveran, 1890, France; Franchini, 1923, Italy. *P.*, Labbé, 1894, France; Schaudinn, quoted by Prowazek, 1911, Germany.

(Alauda cristata) = Galerida cristata (crested lark): *T.*, Sergent, Ed. and Et., 1904, Algeria.

Albanella pallida (falcon): *L. laverani*, Franchini, 1923, Italy.

Amadina erythrocephala (weaver finch): *T.*, Fantham, 1919, South Africa. *P.*, Z.S., 1925, South Africa. *Hg. amadinæ*, Fantham, 1919, 1924, South Africa.

Amadina fasciata (weaver finch): *H.*, Plimmer, 1912, West Africa.

Amblyornis subalaris (bower bird): *H.*, Plimmer, 1912, New Guinea.

Ampelis japonicus (waxwing): *T.*, Ogawa, 1911, Japan.

Anas boscas (mallard or wild duck): *L.*, França, 1912, Portugal.

(Anas moschata) = Cairina moschata (muscovy musk duck): *H.*, Leger, 1918, Guiana.

(Anas querquedula) = Querquedula querquedula (garganey teal): *P* and *L.*, Schaudinn, quoted by Prowazek, 1911, Germany.

Anastomus lamelligerus (open bill): *H.*, Rodhain, Pons, Vandenbranden and Bequaert, 1913, Belgian Congo.

(Andropadus virens) = Eurillas virens (bulbul): *T.*, Zupitza, 1909, Cameroons.

Anellobia chrysoptera (honey-sucker): *T. anellobiæ*, Johnston, 1910, and Cleland and Johnston, 1910, 1911, Australia; *L. anellobiæ*, Cleland and Johnston, 1910, Australia. *H.*, Breinl, 1913, West Australia.

Anorthura troglodytes = (Troglodytes parvulus).

Anser anser = (Anser ferus).

Anser domesticus (goose): *L. anserina*, Knuth, 1922, Germany; Knuth and Magdeburg, 1922, Germany.

(Anser ferus) = Anser anser (grey lag goose): *L.*, França, 1912, Portugal.

Anthus japonicus = (Anthus spinoletta japonicus).

Anthus pratensis (meadow pipit): *T.*, Nieschulz, 1921, 1922, Heligoland.

(Anthus spinoletta japonicus) = Anthus japonicus (pipit): *H.*, Ogawa, 1911, Japan.

Anthus trivialis (tree pipit): *H.*, Galli-Valerio, 1902, Europe.

Antigone antigone (sarus crane): *P.* or *H.*, Z.S., 1925, India.

Aphelocephala leucopsis (whiteface): *H.*, Cleland and Johnston, 1911, Australia.

Aphelocoma sordida (jay): *P.*, Plimmer, 1914, Mexico.

Aptus chopi (hang-nest): *T.* and *Hg.* (*Tx.*) sp., Carini and Maciel, 1916, Brazil.

(Apus apus) = **Cypselus apus** (swift): *T.*, Franchini, 1923, Italy.

Aquila chrysaetus (golden eagle): *H.*, Wülker, 1919, Macedonia.

Aquila sp. (eagle): *H.*, Wülker, 1919, Macedonia.

Ara macao (red and blue macaw): *P.*, Plimmer, 1912, Central America.

(Ardea atricapilla) = **Butorides atricapilla** (green heron): *T.* and *L.*, Leger, A. and M., 1914, Niger.

(Ardea bubulcus) = **Bubulcus lucidus** (buff-backed heron): *T.*, Zupitza, 1909, Cameroons.

(Ardea cærulea) = **Florida cærulea** (heron): *T.*, De Cerqueira, 1906, Brazil.

(Ardea cærulescens) = (?) **Florida cærulea** (heron): *T. ardeæ* var. *major*, Leger, 1918, Guiana.

(Ardea candidissima) = **Leucophoyx candidissima** (egrette): *T.*, De Cerqueira, 1906, Brazil.

Ardea cinerea (heron): *L.*, Wülker, 1919, Macedonia. *T.*, De Cerqueira, 1906, Brazil.

Ardea goliath (goliath heron): *T.*, Rodhain, Pons, Vandenbranden and Bequaert, 1913, Belgian Congo. *L. ardeæ*, Rodhain, Pons, Vandenbranden and Bequaert, 1913, Belgian Congo.

(Ardea purpurea) = **Pyrrherodias purpurea** (purple heron): *H.*, Rodhain, Pons, Vandenbranden and Bequaert, 1913, Belgian Congo; Franchini, 1924, Italy. *L.*, Wülker, 1919, Macedonia; Franchini, 1924, Italy.

(Ardetta flavicollis) = **Dupetor flavicollis** (tiger bittern): *T. chouqueti*, Mathis and Leger, 1911, Tonkin. *H.*, Mathis and Leger, 1910, Tonkin.

Ardetta sinensis (yellow bittern): *T.*, Mathis and Leger, 1911, Tonkin. *H.*, Mathis and Leger, 1910, Tonkin; Ogawa, 1911, Japan. *L. lebœufi*, Mathis and Leger, 1910, Tonkin.

Argusianus argus (Argus pheasant): *T.* and *H.*, Z.S., 1925, Malacca.

Artamus superciliosus (wood swallow): *L.*, Plimmer, 1916, New South Wales.

Asio accipitrinus = **(Otus brachyotus)** (short-eared owl): *H.*, Ziemann, 1898, Heligoland; Leger, 1917, Europe; Celli and San Felici, 1891, Italy.

(Asio leucotis) = **Scops leucotis** : *P.* and *H.*, Z.S., 1925, South Africa.

Asio otus = **(Otus vulgaris** = **Strix otus)** (long-eared owl): *T.*, Nöller, quoted by Nieschulz, 1922, Germany. Böing, 1925, Germany. *H.*, Galli-Valerio, 1902, Europe; Böing, 1925, Germany. *L. danilewskyi*, Ziemann, 1898, Europe; Danilewsky, 1884, Europe; Böing, 1925, Germany.

Astragalinus tristis (American goldfinch): *T. laverani*, Novy and McNeal, 1905, North America.

(Astur badius var. sphenurus) = **Astur sphenurus** (shikra, hawk): *L. martyi*, Commes, 1918, Senegal.

Astur palumbarius (goshawk): *T.*, Mayer, quoted by Nieschulz, 1922, Germany. *H.* and *L.*, Wasielewski, 1908, Germany.

Astur sphenurus = **(Astur badius var. sphenurus).**

Asturinula monogrammica (hawk): *T. asturinulæ*, Stephens and Christophers, 1908; Dutton Todd and Tobey, 1907, Belgian Congo. *H.*, Rodhain, Pons, Vandenbranden, and Bequaert, 1913, Belgian Congo. *L. toddi*, Sambon, 1907, Belgian Congo; Dutton, Todd and Tobey, 1907, Belgian Congo; Aubert and Heckenroth, 1911, Belgian Congo; Rodhain, Pons, Vandenbranden and Bequaert, 1913, Belgian Congo.

Athene brama (Indian little owl): *T. bramæ*, Stephens and Christophers, 1908, India; Donovan, 1904, India. *H.* and *L.*, Donovan (first record), India.

(Athene cuculoides) = **Glaucidium cuculoides** (owl): *T.*, Mathis and Leger, 1911, Tonkin.

Athene noctua (little owl): *T. noctuæ*, Schaudinn, 1904, Europe. Bettencourt and França, 1907, Portugal; Minchin and Woodcock, 1911, Italy; Plimmer, 1914, Europe; Sergent, Ed. and Et., 1904, Algeria; Wülker, 1919, Macedonia. *T.*, Franchini, 1924, Italy. *H. noctuæ*, Celli and San Felici, 1891, Europe; Carda-

matis, 1909, Greece; Plimmer, 1913, Europe; Woodcock, 1914, Italy; Schaudinn, 1904, Italy; Sergent, Ed. and Et., 1904, Algeria; Wülker, 1919, Macedonia; Ziemann, 1898, Italy; Colli and San Felici, 1891, Italy; Grassi and Feletti, 1890, Sicily. *H.*, Schaudinn, quoted by Prowazek, 1911, Germany; Franchini, 1924, Italy. *L. ziemanni*, Laveran, 1902, Europe; Danilewsky, 1890, Europe; Ziemann, 1898, Italy; Schaudinn, 1904, Italy; Moldovan, 1913-1914, Europe; Woodcock, 1912, 1914, Italy; Cardamatis, 1911, Greece; Plimmer, 1913, Europe; Wülker, 1919, Macedonia; Sergent, Ed. and Et., 1907, Algeria. *P.*, Schaudinn, quoted by Prowazek, 1911, Germany. *P. wasielewskii*, Brumpt, 1909, Europe; Sergent, Ed. and Et., 1906, Algeria; Schaudinn, 1904, Italy.

Atticora cyanoleuca (swallow): *P.*, Iturbe and Gonzalez, 1906, Venezuela. *Hg.* (*Tx.*) *atticorœ*, Aragão, 1911, Brazil.

Aythya bæri =(Fuligula bæri).

Balæniceps rex (shoe-billed stork):*L.*, Rodhain, Pons, Vandenbranden and Bequaert, 1913, Belgian Congo.

Balearica ceciliæ (crowned crane): *H.*, Wenyon, 1909, Sudan.

Balearica pavonina (crowned crane): *H.*, Todd and Wolbach, 1912, Gambia.

Balearica regulorum (crowned crane): *H.* and *L.*, Rodhain, Pons, Vandenbranden and Bequaert, 1913, Belgian Congo;*P.*, Plimmer, 1912, South Africa;*P.* and *H.*, Z.S., 1925, South Africa.

Batara cinerea (ant bird): *T.*, Carini and Botelho, 1914, Brazil.

Batara major : *Hg. travassosi*, Raul di Primo, 1925, Brazil.

Biziura lobata (musk duck): *P. biziurœ*, Gilruth, Sweet and Dodd, 1910, Australia.

Botaurus stellaris (bittern): *H.*, Böing, 1925, Germany.

Botaurus sp. (bittern): *L.*, Wülker, 1919, Macedonia.

(Brachyspiza capensis) =Brachyspiza pileata (finch): *P.*, Migone, 1916, Paraguay. *Hg.* (*Tx.*) *brachyspizœ*, Aragão, 1911, Brazil.

Brachyspiza pileata = (Brachyspiza capensis =Zonotrichia pileata).

Bubo bubo (great eagle owl): *H.*, Z.S., 1925, Persia.

Bubo capensis (owl): *H.*, Plimmer, 1912, South Africa.

Bubo lacteus (eagle owl): *H.*, Carpano, 1913, Eritrea; Leger, A. and M., 1914, Niger; Rodhain, Pons, Vandenbranden and Bequaert, 1913, Belgian Congo. *L.*, Leger, A. and M., 1914, Senegal.

Bubo maculosus (eagle owl): *H.*, Plimmer, 1912, 1914, South Africa.

(Bubo pœnsis) =Huhua pœnsis (owl): *H.*, Plimmer, 1912, West Africa.

Bubo sibiricus (Siberian eagle owl): *T.* and *L.*, Böing, 1925, Germany.

Bubo virginianus (eagle owl): *H.*, Galli-Valerio, 1902, Europe.

Bubo sp. (eagle owl): *H.*, Galli-Valerio, 1902, Europe.

Bubulcus lucidus = (Ardea bubulcus).

(Budytes flavus) =Motacilla flava (yellow wagtail): *H.*, Galli-Valerio, 1902, Europe.

Buteo buteo = (Buteo vulgaris).

Buteo desertorum = (Buteo vulpinus).

Buteo lineatus (buzzard): *T. mesnili*, Novy and McNeal, 1905, North America.

(Buteo vulgaris) =Buteo buteo (common buzzard): *H.*, Pfeiffer, 1890, Europe.

(Buteo vulpinus) =Buteo desertorum (buzzard): *H.*, Danilewsky, 1889, South Russia.

Butorides atricapilla = (Ardea atricapilla) (green heron): *T.* and *L.*, Rodhain, Pons, Vandenbranden and Bequaert, 1913, Belgian Congo.

Butorides striata (green heron): *T.*, Leger, 1918, Guiana; Migone, 1916, Paraguay.

Bycanistes albotibialis (hornbill): *T.*, Ringenbach, 1914, Congo.

Bycanistes buccinator (hornbill): *T. bycanistis*, Stephens and Christophers, 1908; Dutton, Todd and Tobey, 1907, Belgian Congo.

Bycanistes cristatus (hornbill): *T.*, Ross, P. H., 1911, East Africa.

(Bycanistes leucopigius) =Bycanistes sharpei (hornbill): *T.*, Rodhain, Pons, Vandenbranden and Bequaert, 1913, Belgian Congo.

Bycanistes sharpei = (Bycanistes leucopigius).

Bycanistes subquadratus (hornbill): *T.*, Minchin, 1910, Uganda.

Caccabis chukar (chukar, red-legged partridge): *T.*, Plimmer, 1912, India.

Caccabis melanocephala (black-headed, red-legged partridge): *H.*, Plimmer, 1915, Arabia.

Caccabis petrosa (Barbary partridge): *H.*, Carpano, 1913, Eritrea.

Caccabis rufa = (Perdix rubra) (common red-legged partridge): *L.*, Schaudinn, quoted by Prowazek, 1911, Zoological Gardens, Berlin.

(Cacicus chrysopterus) = Agelæus thilius (hang-nest): *T.*, Carini and Maciel, 1916, Brazil.

Cairina moschata = (Anas moschata).

(Calliste cyanoptera) = Calospiza cyanoptera (tanager): *H.*, Plimmer, 1912, South America; Iturbe and Gonzalez, 1916, Venezuela.

(Calliste festiva) = Calospiza cyanocephala (tanager): *H.*, Plimmer, 1912, Brazil.

(Calliste melanonota) = Calospiza melanonota (tanager): *H.*, Plimmer, 1912, Brazil.

(Calliste thoracica) = Calospiza thoracica (tanager): *P.*, Plimmer, 1912, Brazil.

(Calliste tricolor) = Calospiza tricolor (tanager): *H.*, Plimmer, 1912, South America.

Calœnas nicobarica (Nicobar pigeon): *H.*, Z.S., 1925, India.

Calopelia puella (bronze-wing pigeon): *H.*, Plimmer, 1912, West Africa.

Calospiza cyanocephala = (Calliste festiva).

Calospiza cyanoptera = (Calliste cyanoptera).

Calospiza fatuosa (superb tanager): *P.*, Z.S., 1925, Brazil.

Calospiza melanonota = (Calliste melanonota).

Calospiza thoracica = (Calliste thoracica).

Calospiza tricolor = (Calliste tricolor) (tanager): *H.*, Carini and Maciel, 1916, Paraguay.

Caprimulgus europæus (nightjar): *T. thiersi*, Leger, 1913, Corsica. *T.*, *H.*, *L.*, and *P.*, Böing, 1925, Germany.

Caprimulgus fossei (nightjar): *T. caprimulgi*, Kérandel, 1909, French Congo. *H.* and *L. caprimulgi*, Kérandel, 1909, 1913, French Congo.

Caprimulgus sp. (nightjar): *H.*, Sergent, Ed. and Et., 1904, Algeria.

Capsiempis flaveola (tyrant bird): *H.*, Iturbe and Gonzalez, 1916, Venezuela.

Cardinalis cardinalis = (Cardinalis virginianus).

Cardinalis phœniceus (finch): *H.*, Plimmer, 1917, Venezuela.

(Cardinalis virginianus) = Cardinalis cardinalis (cardinal): *P.*, Schaudinn, quoted by Prowazek, 1911, Zoological Gardens, Berlin.

Carduelis carduelis = (Carduelis elegans = Fringilla carduelis) (goldfinch): *T.*, Sergent, 1910, Algeria; Cardamatis, 1911, Greece.

(Carduelis elegans) = Carduelis carduelis (goldfinch): *T.*, Plimmer, 1914, Europe. *H.*, Galli-Valerio, 1902, Europe. Cardamatis, 1909, Greece. *P.*, Plimmer, 1914, Europe.

Cariama cristata: *H.*, Lutz and Meyer (quoted by Pinto, 1925), Brazil.

Carpodacus mexicanus (rose finch): *P.*, Plimmer, 1912, Mexico.

Carpophaga concinna (fruit pigeon): *P.*, Plimmer, 1912, Aru Isles; Z.S., 1925, Malaya. *Tx.*, Plimmer, 1915, Aru Isles.

Casarca tadornoides = (Tadorna tadornoides).

Cassiculus melanicterus (hang-nest): *H.*, Plimmer, 1912, Mexico.

(Catharista atratus) = Cathartes urubu (black vulture): *T. catharistæ*, Mesnil, 1912; Brimont, 1909 Guiana. *Hg. pintoi*, Raul di Primio, 1925, Brazil.

Cathartes aura (turkey vulture): *H.*, Darling, 1912, Panama. *Hg. pintoi*, Raul di Primio, 1925, Brazil.

Cathartes burrovianus (South American turkey vulture): *H.*, Z.S., 1925, Brazil.

Cathartes urubu = (Catharista atratus).

Catheturus lathami (mound-builder): *H.*, Cleland and Johnston, 1911, Australia.

Centropus burchelli (cuckoo): *L. centropi*, Fantham, 1921, South Africa.

Centropus monachus (cuckoo): *H.*, Leger, A. and M., 1914, Niger. *L.*, Leger, A. and M., 1914, Senegal.

Centropus senegalensis (cuckoo): *L.*, Aubert and Heckenroth, 1911, French Congo.

Centropus sinensis (cuckoo): *H.*, Mathis and Leger, 1911, Tonkin; Donovan (first record), India. *L.*, Mathis and Leger, 1911, Tonkin.

Centropus superciliosus (cuckoo): *H.*, Minchin, 1910, Uganda; Rodhain, Pons, Vandenbranden and Bequaert, 1913, Belgian Congo. *L.*, Rodhain, Pons, Vandenbranden and Bequaert, 1913, Belgian Congo.

Cephalophoneus bucephalus = (Lanius bucephalus).

Cephalophoneus schach = (Lanius schach).

Cerchneis alopex (kestrel): *H.*, Leger, A. and M., 1914, Niger.

Cerchneis naumanni = (Tinnunculus cenchris).

Cerchneis sparveria = (Falco sparverius).

Cerchneis tinnunculus = (Falco tinnunculus = Tinnunculus alaudarius = Tinnunculus tinnunculus) (common kestrel): *T.*, Böing, 1925, Germany. *H.*, Ziemann, 1898, Heligoland and Italy. *H. danilewskyi* var. *tinnunculus*, Wasielewski and Wülker, 1918, Europe. *H.* and *L.*, Böing, 1925, Germany.

Ceuthmochares æreus (cuckoo): *T.*, Martin, Lebœuf and Roubaud, 1909, Congo; Roubaud, 1909, French Congo.

Chalcomitra amethystina (black sun bird): *H.*, Z.S., 1925, South Africa.

Chalcomitra senegalensis = (Cinnyris senegalensis).

Chamæpelia minuta (grey ground dove): *P.* or *H.*, Z.S., 1925, West Indies.

Chamæpelia talpacoti = (Columbina talpacoti = Columbogallina talpacola) (dove).

Chamæza brevicauda (ant bird): *T.*, Carini and Maciel, 1916, Brazil.

(**Chanthornus jamaicai**) = Icterus jamaicai (hang-nest): *H.*, Aragão, 1916, Brazil.

Chelidon urbica (martin): *T. mathisi*, Sergent, 1904 and 1907, Algeria; Petrie, 1905, England. *H.*, Franchini, 1923, Italy.

(**Chenalopex ægyptiaca**) = Alapochen ægyptiacus ("Egyptian goose," duck): *L.*, Minchin, 1911, Uganda.

(**Chibia bracteata**) = Dicruropsis bracteata (drongo): *H.*, Breinl, 1913, West Australia.

Chlamydodera orientalis (bower bird): *T.* and *H.*, Breinl, 1913, West Australia.

(**Chloris chloris**) = Ligurinus chloris (greenfinch): *P.*, Wasielewski, 1902, Germany; Ziemann, 1898, Italy; Franchini, 1924, Italy.

(**Chloris hortensis**) = Ligurinus chloris (greenfinch): *H.*, Laveran, 1907, France.

Chlorophonia pretrei (tanager): *H.*, Iturbe and Gonzalez, 1916, Venezuela.

Chloropis aurifrons (golden-headed chloropis): *P. præcox* and *H.*, Z.S., 1925, India.

Chlorotreron iozona = (Ptilinopus iozonus).

(**Chroicocephalus ridibundus**) = Larus ridibundus (black-headed gull): *H.*, Franchini, 1924, Italy.

Chrysococcyx cupreus (golden cuckoo): *T.*, Zupitza, 1909, Cameroons.

(**Chrysomitris cucullatus**) = Spinus cucullatus (hooded siskin): H., Plimmer, 1912, North America.

(**Chrysomitris spinus**) = Spinus spinus (siskin): *H.*, Plimmer, 1914, North Europe. *Tx.*, Mayer, quoted by Nöller, 1920, Hamburg; Walzberg, 1923, Berlin.

Cicinnurus regius (king bird of paradise): *T.*, Plimmer, 1915, Aru Isles. *H.*, Plimmer, 1912, New Guinea.

Cinnyris chloropygius (sun bird): *T.*, Leger, A. and M., 1914, Niger.

(**Cinnyris senegalensis**) = Chalocomitra senegalensis (Senegal sun bird): *H.*, Commes, 1918, Niger.

Circaëtus cinereus = (Falco circaëtus).

Circaëtus gallicus (harrier eagle): *L. circaëti*, Sergent and Fabiani, 1922, Algeria.

Circus æruginosus (marsh harrier): *T.*, Böing, 1925, Germany. *H.*, Danilewsky, 1889, South Russia; Franchini, 1924, Italy; Böing, 1925, Germany. *L.*, Franchini, 1924, Italy; Böing, 1925, Germany.

Cittocincla macrura (shama): *T.*, Plimmer, 1914, India. *H.*, Plimmer, 1914 and 1917, and Z.S., 1925, India.

Clivicola riparia =(Cotyle riparia).

Coccothraustes coccothraustes =(Coccothraustes vulgaris) (hawfinch): *P.* and *L.*, Franchini, 1924, Italy. *T.*, *H.*, *L.*, and *P.*, Böing, 1925, Germany.

Coccothraustes japonicus =(Coccothraustes vulgaris japonicus).

(Coccothraustes melanura) =Eophona melanura (hawfinch): *P.*, Plimmer, 1912, Japan.

(Coccothraustes vulgaris) =Coccothraustes coccothraustes (common hawfinch): *T.*, Bettencourt and França, 1907, Portugal. *H.*, Sergent, Ed. and Et., 1904, Algeria (?); Danilewsky, 1889, South Russia. *P.*, Ziemann, 1898, Italy.

(Coccothraustes vulgaris japonicus) =Coccothraustes japonicus (Japanese hawfinch): *T.*, *H.*, and *L.*, Ogawa, 1911, Japan.

(Cœreba cyanea) =Cyanerpes cyaneus (American creeper): *H.*, Plimmer, 1912 and 1913, South America.

Colæus monedula =(Corvus monedula =Monedula monedula = Monedula turrium) (jackdaw): *H.*, Galli-Valerio, 1902, Europe.

Colaptes auratus (woodpecker): *T.*, Novy and McNeal, 1905, North America.

Colaptes campestris (woodpecker): *P.*, Carini and Maciel, 1916, Paraguay.

Coliostruthus laticauda =(Penthetria laticauda).

(Columba domestica) =Columba livia (pigeon, domestic variety): *H.*, Cardamatis, 1909, Greece. *H.*, Galli-Valerio, 1902, Europe.

Columba livia (rock pigeon): *T. hannai*, Pittaluga, 1904 (Hanna, 1903, India). *H.*, Schaudinn, quoted by Prowazek, 1911, Europe; Rodhain, Pons, Vandenbranden and Bequaert, 1913, Belgian Congo; Sergent, Ed. and Et., 1904, Algeria; Celli and San Felici, 1891, Italy. *P.*, Sergent, Ed. and Et., 1904, Algeria.

Columba livia = (Columba domestica).

Columba palumbus (wood pigeon): *T.*, *H.*, *L.*, and *P.*, Böing, 1925, Germany.

Columba rufina (pigeon): *H.*, Leger, 1918, Guiana.

Columba sp. (pigeon): *H. columbœ*, Celli and San Felici, 1891, Europe.

(Columbina talpacoti) =Chamœpelia talpacoti (dove): *H.*, Iturbe and Gonzalez, 1916, Venezuela.

(Columbigallina talpacota) =Chamœpelia talpacoti (dove): *H.*, Aragão, 1916, Brazil.

Copyschus saularis (magpie robin): *T.*, Plimmer, 1912, India; Plimmer, 1913, India. *H. moruoni*, Mello and Braz de Sa, 1916, India. *H.*, Mathis and Leger, 1910, Tonkin; Plimmer, 1912 and 1916, India.

Coracias abyssinicus (roller): *H.*, Carpano, 1913, Eritrea; Leger, A. and M., 1914, Niger; Z.S., 1925, Abyssinia. *L.*, Leger, A. and M., 1914, Senegal.

Coracias garrulus (roller): *T. avium*, Danilewsky, 1885, South Russia. *H.*, Cardamatis, 1909, Greece; Wülker, 1919, Macedonia; Danilewsky, 1889, South Russia. *L.*, Wülker, 1919, Macedonia.

Coracias indica (Indian roller): *H.*, Plimmer, 1912 and 1914, India.

Corcorax melanorhamphus (crow): *L. annellobiœ*, Cleland and Johnston, 1911, Australia.

Corvinella corvina (shrike): *H.* and *L.*, Leger, A. and M., 1914, Senegal.

(Corvus americanus) =Corvus brachyrhynchus (crow): *H.*, Opie, 1898, North America; Galli-Valerio, 1902, Europe.

Corvus brachyrhynchus =(Corvus americanus).

Corvus corax =(Corvus corvus) (raven): *H.*, Galli-Valerio, 1902, Europe. *L. sakharoffi*, Sambon, 1908, Europe; Sakharoff, 1893, Transcaucasia; Leger, 1917, Europe. *P.*, Plimmer, 1913, Europe.

Corvus cornix (grey crow): *H. danilewskyi*, Kruse, 1890, Europe; Wülker, 1919, Macedonia; Kruse, 1890, Italy.

Corvus corone (carrion crow): *H.*, Leger, 1913, Corsica. *L. zuccarellii*, Leger, 1913, Corsica. *L.*, Plimmer, 1917, Europe.

(**Corvus corvus**) = **Corvus corax** (raven): *P.*, Danielewsky, 1898, South Russia (?).

(**Corvus frugilegus**) = **Trypanocorax frugilegus** (rook): *H.*, Danilewsky, 1898, South Russia (?). *L.*, Wasielewski, 1908, Germany; Cardamatis, 1911, Greece.

Corvus japonensis = (**Corvus macrorhynchus japonensis**).

Corvus macrorhynchus (crow): *H.*, Castellani and Willey, 1905, Ceylon, quoted by Dobell, 1910.

(**Corvus macrorhynchus japonensis**) = **Corvus japonensis** (crow): *T.*, Mine, 1914, Japan. *H.*, Mine, 1914, Japan; Ogawa, 1911, Japan. *L.*, Ogawa, 1911, Japan.

(**Corvus monedula**) = **Coloeus monedula** (jackdaw): *L.*, Chingareva (Schingareff), 1911, Russia. *T. corvi*, Stephens and Christophers, 1908, India.

Corvus splendens (Indian house crow): *T.* and *P.*, Donovan (first record), India. *H.*, Castellani and Willey, 1905, Ceylon; Donovan (first record), India.

Corythœola cristata (turaco): *T.*, Minchin, 1910, Uganda. *H.*, Minchin, 1910, Uganda. •

Coturnix communis (quail): *T.*, Franchini, 1924, Italy.

Coturnix delegorguei (quail): *H.*, Rodhain, Pons, Vandenbranden and Bequaert, 1913, Belgian Congo.

(**Cotyle riparia**) = **Clivicola riparia** (sand martin): *T. cotyle*, Franchini, 1923, Italy.

Cracticus destructor (shrike): *H.*, Breinl, 1913, West Australia.

Crateropus striatus (babbler): *H.*, Castellani and Willey, 1905, Ceylon.

(**Crax sclateri**) = **Penelope sclateri** (Sclater's curassow): *T. pedrozi*, Carini and Botelho, 1914, Brazil.

(**Crithagra chrysopyga**) = **Serinus icterus** (serin): *H.*, Plimmer, 1915, West Africa.

(**Crithagra musica**) = **Poliospiza leucopygia** (white-rumped canary): *P.*, Z.S., 1925, West Africa.

Crithagra sp.: *T.*, Dutton and Todd, 1903, Gambia.

Crotophaga ani (cuckoo): *H.*, Iturbe and Gonzalez, 1916, Venezuela.

Cryptorhina afra (Senegal magpie): *P.* or *H.*, Z.S., 1925, West Africa.

Crypturus cinereus (tinamou): *T. tinami*, Mesnil, 1912 (Brimont, 1912, Guiana).

Crypturus obsoletus: *H.*, Lutz and Meyer (quoted by Pinto, 1925), Brazil.

Curæus aterrinus (hang-nest): *L.*, Plimmer, 1915, Chili.

Cyanerpes cyaneus = (**Cœreba cyanea**).

Cyanistes cæruleus = (**Parus cæruleus**).

Cyanocitta cristata (jay): *T. confusum*, Lühe, 1906 (*T. avium*, Novy and McNeal, 1905, North America).

Cyanocorax crysops ?: *H.*, Lutz and Meyer (quoted by Pinto, 1925), Brazil.

Cyanops flavifrons (barbet): *H.*, Plimmer, 1916, Ceylon. *P.*, Plimmer, 1914, Ceylon.

(**Cyanospiza ciris**) = **Passerina ciris** (painted bunting): *L.*, Plimmer, 1916, North America.

(**Cyanospiza leclancheri**) = **Passerina leclancheri** (bunting): *P.*, Plimmer, 1912, Mexico.

Cygnus melanocoryphus = (**Cygnus nigricollis**).

(**Cygnus nigricollis**) = **Cygnus melanocoryphus** (black-necked swan): *P.*, Schaudinn, quoted by Prowazek, 1911, Zoological Gardens, Berlin.

Cypselus apus = (**Apus apus**) (swift): *T. cypseli*, Franchini, 1923, Italy; Rudovsky, 1923, Europe.

Dacelo gigas (kingfisher, laughing jackass): *H.*, Johnston, 1910, Australia; Welsh and Priestley, 1911, Australia.

Dacnis cyana (American creeper): *P.*, Plimmer, 1912, South America.

Dendrocitta rufa = (**Dendrocitta vagabunda**).

(**Dendrocitta vagabunda**) = **Dendrocitta rufa** (magpie): *T.* and *H.*, Plimmer, 1913, India.

Dendrocopus villosus = (**Dryobates villosus**).

Dicæum hirundinaceum (flower pecker): *H.*, Cleland, 1915, Australia.

Dicruropsis bracteata = (**Chibia bracteata**).

(Dryobates villosus) =Dendrocopus villosus (hairy woodpecker): *T.*, Novy and McNeal, 1905, North America.

Duck : *L. anatis*, Wickware, 1915, Canada.

Dupetor flavicollis =(Ardetta flavicollis).

Eclectus pectoralis (red-sided parrot): *H.*, Plimmer, 1912, New Guinea.

Elainea albiceps (tyrant bird): *Hg.* (*Tx.*) sp., Carini and Maciel, 1916, Brazil.

Elanus cœruleus (kite): *T.*, Bettencourt and França, 1907, Portugal.

Elanus hypoleucus =Falco hypoleucus.

Emberiza cia (bunting): *H.*, Galli-Valerio, 1902, Europe.

Emberiza cirlus (cirl bunting): *H.*, Schaudinn, quoted by Prowazek, 1911, Italy. *L. cambournaci*, França, 1912, Portugal.

Emberiza citrinella (yellow-hammer): *T.*, Petrie, 1905, England. *P.* and *H.*, Wasielewski, 1908, Germany.

Emberiza elegans (bunting): *H.*, Ogawa, 1911, Japan.

Emberiza fucata (bunting): *P.*, Plimmer, 1913, India.

Emberiza melanocephala (bunting): *H.*, Cardamatis, 1909, Greece.

(Emberiza projer) =Miliaria miliaria (common bunting): *H.*, Galli-Valerio, 1902, Europe. *P.*, Wasielewski, 1902, Germany.

(Emberiza variabilis) =Tisa variabilis (bunting): *L.*, Ogawa, 1911, Japan. *P.*, Ogawa, 1912, Japan.

Enneoctonus collurio =(Lanius collurio).

Entomyza cyanotis (honey-sucker): *T. anellobiæ*, *H.* and *L. anellobiæ*, Cleland and Johnston, 1911, Australia.

Eophona melanura =(Coccothraustes melanura).

(Eos ricinata) =Eos variegata (lory): *H.*, Plimmer, 1915, Aru Isles.

Eos variegata =(Eos ricinata).

Ephippiorhynchus senegalensis (saddle-billed stork): *H.*, Wenyon, 1909, Sudan.

(Erithacus luscinia) =Ædon megarhyncha (nightingale): *H.*, Ziemann, 1898, Italy.

(Erithacus phœnicurus) =Phœnicurus phœnicurus (redstart): *T.*, Nieschulz, 1921, 1922, Heligoland.

Erithacus rubecula =(Sylvia rubecula) (robin): *T.*, Bettencourt and França, 1907, Portugal; Nieschulz, 1921, 1922, Heligoland. *H.*, Wülker, 1919, Macedonia.

(Erythrocnema unicincta) =Parabutes unicinctus (buzzard): *H.*, Iturbe and Gonzalez, 1916, Venezuela.

Erythropus vespertinus (red-footed falcon): *H.*, Danilewsky, 1889, South Russia.

Erythrura prasina (weaver finch): *H.*, Plimmer, 1912, Sumatra.

Erythrura psittacea (weaver finch): *H.*, Plimmer, 1912, New Caledonia.

Estrilda angolensis (weaver finch): *T.*, Fantham, 1919, South Africa.

Estrilda cinerea (weaver finch): *H.*, Leger, A. and M., 1914, Niger. *P.* and *H.*, Z.S., 1925, West Africa.

Estrilda estrilda (weaver finch): *T. johnstoni*, Dutton and Todd, 1903, Gambia.

(Estrilda melpoda) =sporæginthus melpodus (waxbill): *T.*, Plimmer, 1912, Australia; and *P.*, Plimmer, 1912, West Africa.

(Estrilda phœnicotis) =Uræginthus phœnicotis (waxbill): *P.*, Marullaz, 1912, Africa; *P.* and *L.*, Z.S., 1925, Gambia.

Eudynamis cyanocephala (cuckoo): *H.*, Breinl, 1913, West Australia.

Euethia canora =(Phonipara canora).

Eulabes intermedia =(Gracula intermedia).

Eulabes javanensis (Malay grackle): *H.*, Z.S., 1925, Malacca.

Eulabes religiosa =(Gracula religiosa) : *P.*, Z.S., 1925, South India.

Eunetta falcata (falcated teal): *L.*, Plimmer, 1914, Siberia. *P.*, Plimmer, 1915, Siberia.

Euphagus carolinus =(Scolecophagus carolinus).

Euphonia violacea (tanager): *H.* and *P.*, Iturbe and Gonzalez, 1916, Venezuela.

(Euplectes orix) =Pyromelana orix (weaver finch): *H.*, Plimmer, 1912, West Africa.

Eupodotis arabs (Sudan paauw): *H.*, Z.S., 1925, Sudan.

Eurillas virens = (Andropadus virens).

Eurystomus afer (roller): *T.* and *L.*, Zupitza, 1909, Cameroons.

Eurystomus gularis (roller): *T. eurystomi*, Kérandel, 1909, 1912, French Congo. *L. eurystomi*, Kérandel, 1909, 1913, French Congo.

Euxenura maguari (stork): *H.*, Carini and Maciel, 1916, Paraguay.

Excalfactoria chinensis (Chinese-painted quail): *H.*, Z.S., 1925, China.

(Falco circaëtus) =Circaëtus cinereus (serpent eagle): *H.* and *L.*, Rodhain, Pons, Vandenbranden and Bequaert, 1913, Belgian Congo.

Falco hypoleucus = Elanus hypoleucus (falcon): *T.*, Breinl, 1913, West Australia. *P.*, Breinl, 1912, West Australia.

(Falco nisus) = Accipiter nisus (sparrow hawk): *L.*, Singareva (Schingaroff), 1910, Russia.

Falco peregrinus (peregrine falcon): *L.*, Ogawa, 1911, Japan.

(Falco sparverius) =Cerchneis sparveria (American kestrel): *H.*, Leger, 1918, Guiana.

(Falco tinnunculus) = Cerchneis tinnunculus (kestrel): *T.*, Wasielewski, 1908, Germany. *P.*, Schaudinn, quoted by Prowazek, 1911, Germany. *H.*, Wasielewski, 1908, Germany; Schaudinn, quoted by Prowazek, 1911, Germany; Wülker, 1919, Macedonia; Grassi and Feletti, 1890, Sicily; Pfeiffer, 1890. Germany; Franchini, 1923, Italy. *L.*, Sergent, Ed. and Et., 1902, Algeria.

Falco sp. (falcon): *H.*, Wülker, 1919, Macedonia. *L.*, Donovan (first record), India. *Lamblia sanguinis*, Gonder, 1910, South Africa.

Florida cærulea = (Ardea cærulea and [?] Ardea cærulescens).

Foudia madagascariensis (weaver finch): *T.*, Maya and David, 1912, Mauritius. *H.*, Plimmer, 1913, Madagascar; Maya and David, 1912, Mauritius. *L.*, Maya and David, 1912, Mauritius.

Francolinus bicalcaratus (francolin): *T. francolini*, Kérandel, 1912, French Congo; Leger, A. and M., 1914, Niger. *L. francolini*, Kérandel, 1909, 1913, French Congo; Leger, A. and M., 1914, Senegal.

Francolinus gariepensis (francolin): *T.*, Plimmer, 1912, North Africa.

Francolinus hubbardi (francolin): *T.* and *L.*, Ross, P. H., 1911, East Africa.

Francolinus levaillanti (francolin): *T.* and *L.*, Plimmer, 1912, South Africa.

Francolinus mulemæ (francolin): *H.* and *L.*, Minchin, 1910, Uganda.

Francolinus schuetti = (Francolinus uluensis schuetti).

Francolinus sinensis (francolin): *L. mesnili* and *L. kerandeli*, Mathis and Leger, 1909, Tonkin.

(Francolinus uluensis schuetti) = Francolinus schuetti (francolin): *T.* and *L.*, Ross, P. H., 1911, East Africa.

Francolinus sp. (francolin): *T.*, Todd and Wolbach, 1912, Gambia. *H.*, Rodhain, Pons, Vandenbranden and Bequaert, 1913, Belgian Congo. *L.*, Wenyon, 1909, Sudan; Todd and Wolbach, 1912, Gambia; Rodhain, Pons, Vandenbranden and Bequaert, 1913, Belgian Congo.

(Fringilla cannabina) = Linaria cannabina (linnet): *H.*, Wasielewski, 1908, Germany; Cardamatis, 1909, Greece.

(Fringilla carduelis) = Carduelis carduelis (goldfinch): *T.* and *H.*, Sergent, Ed. and Et., 1904, Algeria. *P.*, Sergent, Ed. and Et., 1904, Algeria; Koch, 1899, Italy; Wasielewski, 1908, Germany.

(Fringilla citrinella) =Spinus citrinellus (siskin): *P.*, Schaudinn, quoted by Prowazek, 1911, Italy.

Fringilla cœlebs (chaffinch): *T. fringillinarum*, Woodcock, 1910, England; Ziemann, 1898, Heligoland; Bettencourt and França, 1907, Portugal; Petrie, 1905, England. *H. fringillæ*, Labbé, 1894, Europe. *H.*, Ziemann, 1898, Heligoland; Cardamatis, 1909, Greece; Woodcock, 1909, England; Wasielewski, 1896 and 1908, Germany; Labbé, 1894, France; Grassi and Feletti, 1890, Sicily; Laveran, 1890, France. *L. fringillinarum*, Woodcock, 1910, England;

Schaudinn, quoted by Prowazek, 1911, Berlin. *P.*, Wasielewski, 1902 and 1908, Germany; Labbé, 1894, France.

(Fringilla kawahahiba minor) =Ligurinus minor (Japanese greenfinch): *H.* and *L.*, Ogawa, 1911, Japan.

(Fringilla linota) =Linaria cannabina (linnet): *T.*, *H.*, and *P.*, Sergent, Ed. and Et., 1904, Algeria.

Fringilla montifringilla (brambling): *L.*, Z.S., 1925, Britain.

(Fringilla petronia) =Petronia petronia (rock sparrow): *T. laverani*, Leger, 1913, Corsica. *H.*, Leger, 1913, Corsica; Cardamatis, 1909, Greece. *L. gentili*, Leger, 1913, Corsica.

(Fuligula bæri) =Aythya bæri (Baer's pochard): *H.*, Plimmer, 1912, India.

Fuligula marila (scaup duck): *L.*, Plimmer, 1912, Europe.

Galerida cristata = (Alauda cristata).

(Gallinago cœlestis) =Gallinago gallinago (snipe): *H.*, Wülker, 1919, Macedonia.

Gallinago gallinago =(Gallinago cœlestis) (snipe): *H.*, Franchini, 1924, Italy.

Gallinula chloropus (moor-hen): *L.*, Coles, 1914, England.

(Gallus bankiva) =Gallus gallus (jungle fowl): *L. schoutedeni*, Rodhain, Pons, Vandenbranden and Bequaert, 1913, Belgian Congo.

(Gallus domesticus) =Gallus gallus (fowl, domestic variety): *T. calmettei*, Mathis and Leger, 1909, Tonkin. *T. gallinarum*, Bruce and Coles, 1911, Uganda; Duke and Robertson, 1912, Uganda. *T.*, Prowazek, 1912, Sumatra. *L. caulleryi* and *L. sabrazesi*, Mathis and Leger, 1909, Tonkin. *L. schüffneri*, Prowazek, 1912, Sumatra. *Hæmotrichomonas gallinarum*, Martoglio, 1917, Eritrea.

(Gallus ferrugineus) =Gallus gallus (jungle fowl): *L.*, Mathis and Leger, 1911, Tonkin.

Gallus gallus =(Gallus bankiva = Gallus domesticus = Gallus ferrugineus) (fowl): *H.*, Plimmer, 1913, Malay.

Gallus varius (jungle fowl): *P.* or *H.*, Z.S., 1925, West Africa.

Garrulax albigularis (babbler): *H.*, Plimmer, 1913, India.

Garrulax leucolophus (babbler): *P.*, Plimmer, 1912, North India.

Garrulus glandarius (common jay): *T.*, Bettencourt and França, 1907, Portugal; Coles, 1914, England. *H.*, Coles, 1914, England; Wasielewski, 1886, Germany; Wülker, 1919, Macedonia; Danilewsky, 1898, South Russia (?); Labbé, 1894, France (?); Laveran, 1890, France. *L. laverani*, França, 1912, Portugal; Coles, 1914, England; Wülker, 1919, Macedonia; Franchini, 1924, Italy. *T.*, *H.*, *L.*, and *P.*, Böing, 1925, Germany.

Garrulus japonicus (jay): *T.*, *H.*, and *L.*, Ogawa, 1911, Japan.

(Garrulus lanceolatus) =Laletes lanceolatus (jay): *H.*, Plimmer, 1914, India.

Garzetta garzetta (little egret): *H.*, Franchini, 1924, Italy.

(Geocichla lunulata) =Oreocichla lunulata (thrush): *H. geocichlæ*, Cleland and Johnston, 1909, Australia.

(Geocichla varia) =Oreocichla varia (thrush): *H. geocichlæ*, Cleland and Johnston, 1909, Australia; Ogawa, 1911, Japan. *L.*, Ogawa, 1911, Japan.

Geopelia striata (dove): *T.*, *H.*, and *L.*, Maya and David, 1912, Mauritius.

Geranospizias gracilis (hawk): *H.*, Iturbe and Gonzalez, 1916, Venezuela.

Gerygone albigularis (fly-catcher): *H.*, Cleland, 1915, Australia.

Glareola pratincola (pratincole): *H.*, Plimmer, 1913, India.

Glaucidium cuculoides = (Athene cuculoides).

Glaucidium perlatum (owl): *T.* and *H.*, Leger, A. and M., 1914, Niger.

(Gracula intermedia) =Eulabes intermedia (wattled starling): *H.*, Plimmer, 1916, North India.

(Gracula religiosa) =Eulabes religiosa (wattled starling): *P.*, Plimmer, 1912, India.

Grallaria imperator (ant bird): *T.*, Carini and Botelho, 1914, Brazil.

Grallina picata (magpie lark): *H.*, Cleland and Johnston, 1911, Australia.

Grus japonensis (crane): *H.*, Plimmer, 1912, North China.

Guttera pucherani (guinea-fowl): *T.* and *L.*, Keysselitz and Mayer, 1909, West Africa.

Gymnorhina leuconota (shrike): *H.*, Plimmer, 1912, Australia.

Gymnorhis flavicollis (finch): *H.*, Plimmer, 1913, India.

Gypagus papa (turkey vulture): *Hg.* (*T.x.*) sp., Carini and Maciel, 1916, Brazil.

(Gypogeranus serpentarius) = Serpentarius serpentarius (secretary bird): *H.*, Carpano, 1913, Eritrea. .

(Hadrostomus rufus) = Pachyrhamphus rufus (American chatterer): *H.*, Carini and Maciel, 1916, Paraguay.

Halcyon senegalensis (kingfisher): *T.*, Zupitza, 1909, Cameroons.

Haliaetus leucoryphus (Palla's fishing eagle): *H.*, Z.S., 1925, India.

Haliaëtus vocifer (eagle): *L. audieri*, Laveran and Nattan-Larrier, 1911, French Congo.

Haliaster girrenera (kite): *T.* and *H.*, Breinl, 1913, West Australia.

(Harporhynchus rufus) = Toxostoma rufum (mocking bird): *T.*, Novy and McNeal, 1905, North America.

Hedydipna platyura = (Nectarina platyura).

Hedymela atricapilla = (Muscicapa atricapilla).

(Hedymeles ludovicianus) = Zamelodia ludoviciana (finch): *H.*, Plimmer, 1912, North America.

Helodromas ochropus (green sandpiper): *H.* and *L.*, Franchini, 1924, Italy.

Herodias alba (heron): *H.*, Rodhain, Pons, Vandenbranden and Bequaert, 1913, Belgian Congo.

Heterospizias meridionalis (hawk): *T. guyanense*, Mesnil, 1912; Brimont, 1912, Guiana.

Hirundo rustica (common swallow): *T.*, Petrie, 1905, England; Franchini, 1923, Italy; Z.S., 1925, Europe. *H.*, Galli-Valerio, 1902, Europe; Franchini, 1923, Italy.

Hirundo sp. (swallow): *H. danilewskyi* var. *hirundinis*, Sergent, Ed. and Et., 1905, Algeria.

Houbara macqueeni (bustard): *H.*, Plimmer, 1912, West Asia.

Huhua poensis – (Bubo poensis).

Hylocichla musica = (Turdus musicus = Turdus philomelos).

Hylocichla mustelinus = (Turdus mustelinus).

Hyphantornis cucullatus – (Hyphantornis textor) (weaver-finch): *T.*, *H.*, and *L.*, Leger, A. and M., 1914, Senegal. *P.*, Z.S., 1925, West Africa.

(Hyphantornis melanocephala) = Sitagra melanocephala (weaver bird): *T. bouffardi* and *L. bouffardi*, Leger and Blanchard, 1911, French Sudan. *P.*, Leger and Blanchard, 1911, Senegal.

(Hyphantornis personata) = Sitagra luteola (Lichtenstein's slender-billed weaver) *H.*, Z.S., 1925, West Africa.

Hyphantornis spilonotus (weaver-finch): *H.*, Plimmer, 1912, Africa.

(Hyphantornis tæniopterus) = Sitagra intermedius (weaver bird): *H.*, Wenyon, 1909, Sudan.

(Hyphantornis textor) = Hyphantornis cucullatus (weaver finch): *H.*, Plimmer, 1913, Gambia.

Hypolais icterina = (Sylvia hypolais).

Hypsipetes amaurotis (bulbul): *H.* and *L.*, Ogawa, 1911, Japan.

Ianthocincla rufigularis (rufous-chinned laughing thrush): *L.*, Z.S., 1925, India.

Ibis æthiopica (ibis): *H.*, Minchin, 1910, Uganda.

Icterus baltimore = (Icterus galbula).

(Icterus galbula) = Icterus baltimore (hang-nest): *T. confusum*, Lühe, 1906 (*T. avium*, Novy and McNeal, 1905, North America).

Icterus jamaicai = (Chantornus jamaicai) (hang-nest): *T.*, Plimmer, 1914, Brazil. *P.*, Plimmer, 1912, Brazil.

(Ixus hainanus) = Pycnonotus hainanus (bulbul): *T. brimonti* and *L. brimonti*, Mathis and Leger, 1910, Tonkin.

Iynx torquilla (wryneck): *H.*, Wülker, 1919, Macedonia; Ogawa, 1911, Japan. *H.* and *L.*, Franchini, 1924, Italy.

(Lagonosticta minima) = Lagonosticta senegala (weaver finch): *T.*, Leger, A. and M., 1914, Niger.

Lagonosticta senegala = (Lagonosticta minima) (weaver finch): *T. lagonostictæ*, Marullaz, 1914, Africa. *H.*, Commes, 1918, Niger. *P.*, Marullaz, 1912, Africa.

Lagopus scoticus (grouse): *H. mansoni*, Sambon, 1907, England; Fantham, 1910, England. *L. lovati*, Sambon, 1907, British Isles; Fantham, 1910, British Isles.

Laletes lanceolatus = (Garrulus lanceolatus).

Lamprocolius australis (Burchell's glossy starling): *P. præcox*, Z.S., 1925, South Africa.

Lamprocolius purpureus (tree starling): *H.*, Leger, A. and M., 1914, Niger.

(Lamprotornis æneus) = Lamprotornis caudatus (tree starling): *P.*, Plimmer, 1912, West Africa.

Lamprotornis caudatus = (Lamprotornis æneus).

Lamprotreron superba = (Ptilopus superbus).

(Laniarius cruentus) = Rhodophoneus cruentus (shrike): *T.*, Neave, 1906, Sudan.

(Lanius auriculatus) = Phoneus auriculatus (shrike): *T.*, Leger, A. and M., 1914, Niger.

(Lanius bucephalus) = Cephalophoneus bucephalus (shrike): *T.* and *H.*, Ogawa, 1911, Japan.

(Lanius collurio) = Enneoctonus collurio (red-backed shrike): *T.*, Sjöbring, 1899, Sweden. *H.*, Ziemann, 1898, Heligoland; Wasielewski, 1908, Germany; Wülker, 1919, Macedonia. *T.*, *H.*, *L.*, and *P.*, Böing, 1925, Germany.

Lanius excubitor (great grey shrike): *T.*, Cardamatis, 1910, Greece. *P.*, Schaudinn, quoted by Prowazek, 1911, Germany. *H.*, Danilewsky, 1889, South Russia; Cardamatis, 1909, Greece; Sergent, Ed. and Et., 1904, Algeria; Wülker, 1919, Macedonia. *T.*, *H.*, *L.*, and *P.*, Böing, 1925, Germany.

Lanius minor (shrike): *H.*, Danielewsky, 1889, South Russia; Cardamatis, 1909, Greece.

(Lanius rufus) = Phoneus rutilus (shrike): *H.*, Danilewsky, 1889, South Russia.

(Lanius schach) = Cephalophoneus schach (shrike): *H.*, Mathis and Leger, 1910, Tonkin.

Lanius sp. (shrike): *H.*, Schaudinn, quoted by Prowazek, 1911, Italy; Wülker, 1919, Macedonia. *L.*, Schaudinn, quoted by Prowazek, 1911, Italy.

Larus cachinnans (gull): *H.*, Reichenow, 1913, Europe (?).

Larus cirrhocephalus (gull): *H.*, Thiroux, 1911, Senegal.

Larus ridibundus = (Chroicocephalus ridibundus).

Leistes guianensis (hang-nest): *T.*, Plimmer, 1912, Guiana.

Leptoptilus crumeniferus (marabou stork): *H. crumenium*, Hirst, 1905, and Z.S., 1925, Africa.

Leptoptilus sp. (stork): *H.*, Mathis and Leger, 1910, Tonkin.

Leucophoyx candidissima = (Ardea candidissima).

Leucopternis albicollis = (Urubitinga albicollis).

Ligurinus chloris = (Chloris chloris = Chloris hortensis = Passer chloris) (greenfinch): *Tx.*, Nöller, 1923, Berlin.

Ligurinus minor = (Fringilla kawahahiba minor).

Linaria cannabina = (Fringilla cannabina = Fringilla linota = Linota cannabina).

(Linota cannabina) = Linaria cannabina (linnet): *T.*, Bettencourt and França, 1907, Portugal.

Linota rufescens (finch): *T. fringillinarum*, Woodcock, 1910, England.

Liothrix luteus (babbler): *T. liothricis, L. liothricis,* and *P. tenue,* Laveran and Marullaz, 1914, Japan. *P.,* Z.S., 1925, India. *L.,* Knowles, 1925, India.

Lissotis maculipennis = (Otis maculipennis).

Lissotis melanogaster = (Otis melanogaster) (bustard): *H.,* Todd and Wolbach, 1912, Gambia.

Lophoceros fasciatus (hornbill): *T.,* Zupitza, 1909, Cameroons.

Lophoceros nasutus (hornbill): *T.,* Todd and Wolbach, 1912, Gambia.

Loriculus galgulus (parrot): *T.,* Plimmer, 1913 and 1914, Malay. *H.,* Plimmer, 1912, 1913, 1914, and 1915, Malay and Malacca.

Loriculus indicus (Ceylon loriquet): *H.,* or *P.,* Z.S., 1925, Ceylon.

Lorius domicella (lory): *H.,* Plimmer, 1912, Moluccas.

Lorius flavopalliatus (lory): *H.,* Plimmer, 1917, Batchian.

Loxia curvirostra (crossbill): *T. loxiæ,* Nöller, 1920, Germany. *H.,* Wasielewski, 1908, Germany; Plimmer, 1913, Europe. *P.,* Plimmer, 1913, Europe.

Lullula arborea = (Alauda arborea).

(Luscinia phœnicurus) = Phœnicurus phœnicurus (redstart): *H.,* Sjöbring, 1912 Sweden.

Luscinia sp. (nightingale): *H.* and *P.,* Sergent, Ed. and Et., 1904, Algeria.

Lyrurus testrix (blackgame): *T., H., L.,* and *P.,* Böing, 1925, Germany.

Machlolophus xanthogenys (tit): *H.,* Plimmer, 1912, India.

Macrocorax fuscicapillus (crow): *T.,* Plimmer, 1915, Aru Isles.

Malimbus nitens (Gray's malimbe): *P.* or *H.,* Z.S., 1925, West Africa.

Megalurus gramineus (warbler): *H.,* Breinl, 1913, West Australia.

Melænornis edoloides (fly-catcher): *H.,* Commes, 1918, Niger.

Meleagris gallopavo (turkey, domestic): *H.* sp., Macfie, 1916, West Africa. *L. smithi,* Laveran and Lucet, 1905, Europe.

Melierax gabar (chanting goshawk): *H.* and *L.,* Leger and Husnot, 1912, Senegal.

Meliornis novæ-hollandæ (honey-sucker): *H. meliornis,* Cleland and Johnston, 1909, Australia.

Melithreptes atricapillus (honey-sucker): *H.,* Johnston, 1910, and Cleland and Johnston, 1910, Australia.

Melithreptes validirostris (honey-sucker): H., Cleland, 1915, Australia.

Melittophagus variegatus (bee-eater): *T.,* Zupitza, 1909, Cameroons.

Melopelia leucoptera (dove): *H. melopeliæ,* Laveran and Pettit, 1909, Australia.

Melophus melanicterus (bunting): *H.* and *P.,* Plimmer, 1913, India.

(Melospiza fasciata) = Melospiza melodia (American song sparrow): *T.,* Novy and McNeal, 1905, North America. *H.,* Opie, 1898, North America; Galli-Valerio, 1902, Europe.

Melospiza georgiana (American song sparrow): *H.,* Galli Valerio, 1902, Europe. *P.,* Opie, 1898, North America.

Melospiza melodia = (Melospiza fasciata).

Merops albicollis (bee-eater): *T.* and *H.,* Minchin, 1910, Uganda.

Merops apiaster (European bee-eater): *H.,* Wülker, 1919, Macedonia.

Merops ornatus (bee-eater): *H.,* Cleland and Johnston, 1911, Australia.

Merula albiventer = (Planesticus albiventer).

Merula boulboul (thrush): *P.,* Plimmer, 1915, India.

Merula fumigata = (Turdus fumigatus).

Merula merula = (Merula vulgaris) (blackbird): *T.,* Petrie, 1905, England; França, 1912, Portugal; Coles, 1914, England; Franchini, 1923, Italy. *H.,* Coles, 1914, England. *L. mirandæ,* França, 1912, Portugal; Coles, 1914, England; Wülker, 1919, Macedonia.

(Merula migratoria) = Turdus migratorius (American robin, thrush): *T. confusum,* Lühe, 1906 (*T. avium,* Novy and McNeal, 1905, North America). *P. vaughani,* Novy and McNeal, 1904, North America.

Merula rufiventer =(Turdus rufiventris).

(Merula vulgaris) =Merula merula (blackbird): *T.* and *H.*, Coles, 1914, England.

Mesia argentauris (babbler): *H.*, Plimmer, 1913, India.

Micrœca fascinans (fly-catcher): *T. anellobiœ* and *H.*, Cleland and Johnston, 1911, Australia.

Miliaria miliaria =(Emberiza projer).

Milvago chimachima (carrion hawk): *H.*, Iturbe and Gongalez, 1916, Venezuela.

Milvus ægyptus (kite): *L.*, Neave, 1909, Central Africa.

Milvus govinda (kite): *T. milvi*, Stephens and Christophers, 1908, India; Donovan, quoted by Thiroux, 1905, India.

Milvus korschun =(Milvus migrans).

(Milvus migrans) =Milvus korschun (black kite): *H.*, Danilewsky, 1889, South Russia; Rodhain, Pons, Vandenbranden and Bequaert, 1913, Belgian Congo. *L.*, Rodhain, Pons, Vandenbranden and Bequaert, 1913, Belgian Congo.

Milvus milvus (common kite): *T., H.,* and *L.*, Böing, 1925, Germany.

Mimeta sagittata =(Oriolus sagittarius).

(Monedula monedula) =Colæus monedula (jackdaw): *L.*, Šingareva (Schingareff), 1910, Russia.

(Monedula turrium) =Colæus monedula (jackdaw): *H.*, Danilewsky, 1889, South Russia.

Monticola saxatilis (rock thrush): *H., L.,* and *P.*, Wülker, 1919, Macedonia.

Motacilla alba (white wagtail): *L.*, Franchini, 1924, Italy.

Motacilla flava =(Budytes flava).

Motacilla sp. (wagtail): *T.*, Bettencourt and França, 1907, Portugal.

Munia maja (white-headed mannakin): *P. præcox*, Z.S., 1925, Malacca.

Munia orizivora =(Padda orizivora) : *H.*, Z.S., 1925, Java.

Munia topela (weaver finch): *H.* and *L. roubaudi*, Mathis and Leger, 1910, Tonkin.

(Muscicapa atricapilla) =Hedymela atricapilla (pied fly-catcher): *T.*, Bettencourt and França, 1907, Portugal; Nieschulz, 1921, 1922, Heligoland.

Muscicapa grisola (spotted fly-catcher): *H.*, Cardamatis, 1909, Greece.

Muscicapa sp. (fly-catcher): *H.*, Wülker, 1919, Macedonia.

Muscivora tyrannus (tyrant bird): *H.*, Carini and Maciel, 1916, Paraguay.

Musophaga rossæ (turaco): *H.*, Minchin, 1910, Uganda.

Mycteria americana : *H.*, Lutz and Meyer (quoted by Pinto, 1925), Brazil.

Myiagra nitida (fly-catcher): *H.*, Cleland and Johnston, 1910, and Johnston, 1910, Australia.

Myiozetetes similis =(Myiozetetes texensis).

(Myiozetetes texensis) =Myiozetetes similis (tyrant bird): *H.*, Iturbe and Gonzalez, 1916, Venezuela.

Myzantha garrula (honey-sucker): *H.* and *L. anellobiœ*, Cleland and Johnston, 1911, Australia.

Myzomela sanguineolenta (honey-sucker): *H.* and *L. anellobiœ*, Cleland and Johnston, 1911, Australia.

Necrosyrtes monachus =(Neophron monachus) (hooded vulture): *H.*, Z.S., 1925, Gambia.

(Nectarina platyura) =Hedydipna platyura (sunbird): *H.*, Leger, A. and M., 1914, Niger.

Nectarina sp. (sunbird): *T.*, Zupitza, 1909, Cameroons.

(Neophron monachus) =Necrosyrtes monachus (hooded vulture): *H.*, Todd and Wolbach, 1912, Gambia; Leger, A. and M., 1914, Niger.

Neophron percnopterus (vulture): *T.*, Neave, 1906, Sudan.

Nettion castaneum (teal): *H. nettionis*, Johnston and Cleland, 1909, Australia.

Nettion crecca =(Querquedula crecca).

Nettopus coromandelianus (cotton teal): *H.*, Plimmer, 1915, India.

Ninox boobook (owl): *T.*, Breinl, 1913, West Australia. *H.*, Cleland and Johnston, 1910, Australia. *H. noctuæ*, Breinl, 1913, West Australia.

Ninox strenua (owl): *H.*, Cleland, 1915, Australia.

Notophoyx novæ-hollandiæ (heron): *T.* and *H.*, Breinl, 1913, West Australia.

Nucifraga caryocatactes (nut-cracker): *P.*, Plimmer, 1912, Europe.

Numida meleagris (guinea-fowl): *T. numidæ*, Wenyon, 1909; Kérandel, 1909, French Congo. *H.*, Todd and Wolbach, 1912, Gambia; Kérandel, 1909 and 1913, Congo. *L.*, Kérandel, 1909, French Congo; Franchini, 1924, Italy.

Numida ptilorhyncha (guinea-fowl): *T. numidæ*, Wenyon, 1909, Sudan. *H.*, Minchin, 1910, Uganda; Wenyon, 1909, Sudan; Rodhain, Pons, Vandenbranden and Bequaert, 1913, Belgian Congo. *L. neavei*, Balfour, 1906, Sudan; Minchin, 1910, Uganda; Rodhain, Pons, Vandenbranden and Bequaert, 1913, Belgian Congo; Wenyon, 1909, Sudan. *P.*, Minchin, 1910, Uganda; Wenyon, 1909, Sudan.

Numida sp. (guinea-fowl): *T.*, *P.*, *L.*, and *H.*, Ross, P. H., 1911, East Africa.

Nyctanassa violacea (heron): *T. ardea*, Leger, 1918, Guiana. *H.* (?), Z.S., 1925, America.

(Nycticorax gardenia) = **Nycticorax nycticorax** (night heron): *T. nycticoracis*, Stephens and Christophers, 1908; de Cerqueira, 1906, Brazil; Aragão, 1906, Brazil. *H.*, Lutz and Meyer (quoted by Pinto, 1925), Brazil.

Nycticorax nycticorax = **(Nycticorax gardenia)** (night heron): *H.*, Franchini, 1924, Italy. *L.*, Aubert and Heckenroth, 1911, French Congo; Franchini, 1924, Italy.

Odontophorus capueira: *H.*, Lutz and Meyer (quoted by Pinto, 1925), Brazil.

Onopopelia humilis = **(Turtur humilis)**.

Oreicola ferrea (chat): *L.*, Plimmer, 1913, India.

Oreocichla lunulata = **(Geocichla lunulata)** (thrush): *H.*, Cleland and Johnston, 1909, Australia.

Oreocichla varia = **(Geocichla varia)**.

Oriolus galbula (golden oriole): *H.*, Cardamatis, 1909, Greece. *L.*, Cardamatis, 1911, Greece.

(Oriolus sagittarius) = **Mimeta sagittata** (Australian oriole): *T. anellobiæ*, *H.* and *L. anellobiæ*, Cleland and Johnston, quoted by Johnston, 1912, Australia.

(Orthotomus sutorius) = **Sutoria sutoria** (tailor bird): *T.*, Mathis and Leger, 1911, Tonkin. *H.*, Mathis and Leger, 1910, Tonkin.

Ortygospiza polyzona (weaver-finch): *P.*, Plimmer, 1915, South Africa.

(Otis maculipennis) = **Lissotis maculipennis** (bustard): *P.* and *H.*, Ross, P. H., 1911, East Africa.

(Otis melanogaster) = **Lissotis melanogaster** (bustard): *H.*, Rodhain, Pons, Vandenbranden and Bequaert, 1913, Belgian Congo.

(Otis tetrax) = **Tetrax tetrax** (little bustard): *H.* and *L.*, Wülker, 1919, Macedonia.

Otocompsa emeria = **(Pycnonotus jocosus)**.

(Otus brachyotus) = **Asio accipitrinus** (short-eared owl): *H.* and *L.*, Schaudinn, quoted by Prowazek, 1911, Germany.

(Otus vulgaris) = **Asio otus** (long-eared owl): *H.*, Danilewsky, 1889, South Russia.

Owl: *L.*, Berestneff, 1904, Russia; Lutz and Meyer, 1908, Brazil; Ziemann, 1902, Cameroons.

Pachyrhamphus polychropterus (American chatterer): *H.*, Carini and Maciel, 1916, Paraguay.

Pachyrhamphus rufus = **(Hadrostomus rufus)**.

(Padda orizivora) = **Munia orizivora** (weaver bird, Java sparrow): *T. paddæ*, Laveran and Mesnil, 1904, Asia; Levaditi, 1904, Asia; Thiroux, 1905, Asia; Anschütz, 1910, Asia. *H. oryzivoræ*, Anschütz, 1909 and 1910, Asia; Plimmer, 1915, Java; Thiroux, 1911, Africa; Woodcock, 1910 (?), imported to England; Sergent, Ed. and Et., 1904, imported (?) Algeria. *P.*, Mayer, 1910, Uganda.

Palæornis cyanocephala (parrot): *H.*, Mathis and Leger, 1910, Tonkin.

Palæornis fasciata (parrot): *H.*, Plimmer, 1913, India.

Pandion haliaetus (osprey): *H.*, Danilewsky, 1889, South Russia.

Parabuteo unicinctus = (Erythrocnema unicincta).

(Paradisea rubra) = Uranornis rubra (red bird of paradise): *H.*, Plimmer, 1916, Waigiou Isle.

Pardalotus melanocephalus (flower-pecker): *H.*, Cleland, 1915, Australia.

Paroaria capitata: *Hg. aragãoi*, Raul di Primo, 1925, Brazil.

Paroaria larvata (finch): *Hg. (Tx.) poroariæ*, Aragão, 1911, Brazil.

Parotia lawesi (bird of paradise): *H.*, Plimmer, 1912, New Guinea.

(Parus ater) = Periparus ater (coal tit): *H.*, Galli-Valerio, 1902, Europe.

(Parus cæruleus) = Cyanistes cæruleus (blue tit): *L. majoris*, França, 1912, Portugal. *H.*, Wasielewski, 1908, Germany.

Parus major (great tit): *L. majoris*, Laveran, 1902, France; Wülker, 1919, Macedonia; França, 1912, Portugal. *L.*, Franchini, 1924, Italy. *H.*, Wasielewski, 1908, Germany.

(Parus palustris) = Pœcile palustris (marsh tit): *H.*, Galli-Valerio, 1902, Europe.

(Passer arcuatus) = Passer melanurus (sparrow): *H.*, Plimmer, 1912 and 1913, South Africa.

(Passer chloris) = Ligurinus chloris (greenfinch): *H.*, Sergent, Ed. and Et., 1904, Algeria. *L. scabæ*, França, 1912, Portugal; *P.*, Sergent, Ed. and Et., 1904, Algeria.

Passer domesticus (house sparrow): *T.*, Novy and McNeal, 1905, North America; Bettencourt and França, 1907, Portugal. *T. mayæ*, Maya and David, 1912, Mauritius. *H.*, Opie, 1898, North America; Maya and David, 1912, Mauritius; Wasielewski, 1908, Germany; Cardamatis, 1909, Greece; Schaudinn, quoted by Prowazek, 1911, Italy; Franchini, 1923, Italy; Petrochi and Zuccarini, 1925, Buenos Aires. *L.*, Maya and David, 1912, Mauritius; Ruge, 1901, Germany; Frosch, 1901, Germany. *P.*, Grassi and Feletti, 1891, Europe; Opie, 1898, North America; Petrochi and Zuccarini, 1925, Buenos Aires. *P. passeris*, Johnston and Cleland, 1909, Australia; Koch, 1899, Italy; Schaudinn, quoted by Prowazek, 1911, Germany; Wasielewski, 1902, Germany. *Tx.*, Nöller, 1923, Berlin.

Passer hispaniolensis (Spanish sparrow): *H.*, Grassi and Feletti, 1890, Sicily. *P.*, Grassi and Feletti, 1890, Italy.

Passer italiæ (Italian sparrow): *H.*, Ziemann, 1898, Italy; Celli and San Felici, 1891, Italy; Schaudinn, quoted by Prowazek, 1911, Italy; Franchini, 1924, Italy. *Tx.*, Franchini, 1924, Italy.

Passer melanurus = (Passer arcuatus).

Passer montanus (tree sparrow): *T.*, Mine, 1914, Japan. *H.*, Mine, 1914, Japan; Grassi and Feletti, 1890, Sicily; Franchini, 1924, Italy. *L.*, Mine, 1914, Japan. *P.*, Wasielewski, 1902, Germany; Mine, 1914, Japan. *Tx.*, Franchini, 1924, Italy.

Passer sp. (?) (sparrow): *T.*, Sergent, Ed. and Et., 1904, Algeria. *H. passeris*, Celli and San Felici, 1891, Europe. *H. rouxi*, Novy and McNeal, 1904, North America. *H.*, Sergent, Ed. and Et., 1904, Nigeria; Wenyon, 1911, Bagdad. *P.*, Sergent, Ed. and Et., 1904, Algeria; Mine, 1914, Japan; Ross, R., 1897-1899, India. *Tx.* (?), Adie, 1908, Punjab.

Passerina ciris = (Cyanospiza ciris).

Passerina leclancheri = (Cyanospiza leclancheri).

Pavo cristatus (peacock): *L. martini*, Mathis and Leger, 1911, Tonkin.

Penelope obscura : *H.*, Lutz and Meyer (quoted by Pinto), 1925, Brazil.

Penelope pileata (red-breasted guan): *H.*, Z.S., 1925, South America.

Penelope sclateri = (Crax sclateri).

Penelope superciliaris (guan): *H.*, Carini and Maciel, 1916, Paraguay.

Penthetria albonotata = (Urobrachya albonotata).

(Penthetria laticauda) = Coliostruthus laticauda (weaver finch): *H.*, Plimmer, 1913, East Africa.

(Perdix cinerea) =Perdix perdix (common partridge): *P.*, Laveran and Lucet, 1905, Europe.

Perdix perdix = (Perdix cinerea): *T.*, *H.*, *L.*, and *P.*, Böing, 1925, Germany.

(Perdix rubra) =Caccabis rufa (common red-legged partridge): *L.*, Sergent, Et., 1917, Algeria.

(Pericrocotus elegans) =Pericrocotus speciosus (minivet): *H.*, Mathis and Leger, 1910, Tonkin.

Pericrocotus speciosus = (Pericrocotus elegans).

Periparus ater = (Parus ater).

Pernis apivorus (honey buzzard): *P.*, Danilewsky, 1889, South Russia.

Petrœca phœnicea (fly catcher): *H.*, Cleland and Johnston, 1911, Australia.

Petronia petronia = (Fringilla petronia).

Phalacrocorax africanus (cormorant): *H.*, Rodhain, Pons, Vandenbranden and Bequaert, 1913, Belgian Congo.

Phasianus colchicus (pheasant): *L. macleani*, Sambon, 1908, Europe; *T.*, *H.*, *L.*, and *P.*, Böing, 1925, Germany.

Phasianus mongolicus (pheasant): *P.*, Plimmer, 1914, Mongolia.

(Philemon corniculatus) = Tropidorhynchus corniculatus (honey-sucker): *H. philemon*, Cleland and Johnston, 1909, Australia.

Phoneus auriculatus = (Lanius auriculatus).

Phoneus rutilus = (Lanius rufus).

Phœnicurus auroreus = (Ruticilla aurorea).

Phœnicurus phœnicurus = (Erithacus phœnicurus = Luscinia phœnicurus = Ruticilla phœnicurus).

Phœnicurus titys = (Ruticilla titys).

(Phonipara canora) = Euethia canora (finch): *P.*, Plimmer, 1912, Cuba.

Phylloscopus bonellii (warbler): *H.*, Galli-Valerio, 1902, Europe.

(Phylloscopus collybita) =Phylloscopus rufa (chiffchaff, warbler): *T.*, Bettencourt and França, 1907, Portugal.

Phylloscopus rufa = (Phylloscopus collybita) (chiffchaff, warbler): *H.*, Galli-Valerio, 1902, Europe.

Phylloscopus sibilator = (Sylvia sibilatrix).

Phylloscopus trochilus = (Sylvia trochila) (willow warbler): *T.*, Nieschulz, 1921, 1922, Heligoland.

Piaya cayana = (Piaya cayana macrura).

(Piaya cayana macrura) =Piaya cayana (cuckoo): *T.*, Carini and Botelho, 1914, Brazil.

(Pica caudata) =Pica pica (magpie): *H.*, Danilewsky, 1889, South Russia. *P.*, Kruse, quoted by Minchin, 1903.

(Pica melanoleuca) =Pica pica (magpie): *L.*, Leger, 1917, Europe.

Pica pica = (Pica caudata = Pica rustica = Pica melanoleuca) (magpie): *T.*, Böing, 1925, Germany. *L. berestneffi*, Sambon, 1908, Europe; Sakharoff, 1893. Transcaucasia; Böing, 1925, Germany.

(Pica rustica) =Pica pica (magpie): *L.*, Wülker, 1919, Macedonia.

Pigeon (Columba sp. ?): *T. columbœ*, Stephens and Christophers, 1908, India. *T. hannai*, Mello and Braz de Sa, 1916, India. *H.*, Sergent and Béguet, 1914, Algeria; Acton and Knowles, 1914, India; Adie, 1915, India; Gonder, 1916, South Africa; Mello and Braz de Sa, 1916, India; Aragão, 1907, 1908, 1916, Brazil; Leger, 1918, Guiana; Sergent, Ed. and Et., 1905, Algeria; Negri, 1913, Italy. *P.*, Carini, 1912, Brazil; Whitmore, 1918, New York. *Hœmotrichomonas columbœ*, Lanfranchi, 1917, Europe; Ross, P. H., 1911, East Africa. *Tx. gondii*, Arantes, 1911, Brazil.

Pionus menstrus (red-vented parrot): *H.*, Z.S., 1925, South America.

(Pistrornia scops) =Scops scops (scops owl): *H.*, *L.*, and *Tx.*, Franchini, 1924, Italy.

Pitangus maximiliani = (Pitangus sulphuratus maximiliani).

Pitangus sulphuratus (tyrant bird): *H.*, Plimmer, 1914, South America.   *Hg.* (*Tx.*) sp., Carini and Maciel, 1916, Brazil.

(Pitangus sulphuratus maximiliani) = **Pitangus maximiliani** (tyrant bird): *T.*, Carini and Botelho, 1914, Brazil.   *H.*, Iturbe and Gonzalez, 1916, Venezuela.

Pitta novæ-guineæ (pitta): *P.*, Plimmer, 1917, Aru Isles.

(Planesticus albiventer) = **Merula albiventer** (thrush): *H.*, Iturbe and Gonzalez, 1916, Venezuela.

Platycercus adelaidæ (parrot): *H.*, Cleland and Johnston, 1911, Australia.

Ploceinæ sp. (weaver finch): *T.*, Zupitza, 1909, Cameroons.

Ploceipasser mahali (Smith's sparrow weaver): *P. præcox*, Z.S., 1925, South Africa.

Plotus rufus (darter): *H.*, Rodhain, Pons, Vandenbranden and Bequaert, 1913, Belgian Congo.

Pœcile palustris = (Parus palustris).

Pœocephalus fusicollis (parrot): *H.*, Plimmer, 1912 and 1916, Gambia.

Poëphila gouldiæ (weaver finch): *T.*, Plimmer, 1913, Australia.

Poliospiza leucopygia = (Crithagra musica).

Polyborus tharus (carrion hawk): *H.*, Carini and Maciel, 1916, Paraguay.

Polyplectron bicalcaratum (peacock pheasant): *H.*, Plimmer, 1914, Malay.

Polyplectron germaini (peacock pheasant): *T. polyplectri*, Vassal, 1905, Annam.

Pomatorhinus superciliosus (scimitar babbler): *H.*, Johnston, 1910, and Cleland and Johnston, 1910, Australia.

Poroaria capitata : *Hg.*, aragáoi, Raul di Primo, 1925, Brazil.

(Porphyrio madagascarensis) = **Porphyrio porphyrio** (purple gallinule rail): *H.*, Plimmer, 1912, Madagascar.

Porphyrio porphyrio = (Porphyrio madagascarensis).

Porzana pusilla (Baillon's crake): *H. porzanæ*, Galli-Valerio, 1907, Tunis.

Pratincola caprata (chat): *P.*, Plimmer, 1913, India.   *Tx.*, Plimmer, 1916, India.

Pratincola rubetra (whin-chat): *H.*, Galli-Valerio, 1902, Europe; Ziemann, 1898, Heligoland.

Prionops plumata (wood-shrike): *L.*, Leger, A. and M., 1914, Senegal.

Prionops talacoma (wood-shrike): *T.* and *L.*, Rodhain, Pons, Vandenbranden and Bequaert, 1913, Belgian Congo.

Propasser rhodochrous (finch): *H.* and *L.*, Plimmer, 1917, India.

Pseudotantalus alba (wood-ibis): *H.*, Leger, A. and M., 1914, Niger.

Pseudotantalus ibis (wood-ibis): *H.*, Rodhain, Pons, Vandenbranden and Bequaert, 1913, Belgian Congo.

Psittacus erythacus (grey parrot): *T.*, Zupitza, 1909, Cameroons.

Pternistes infuscatus (bare-throated francolin): *T.* and *L.*, Ross, P. H., 1911, East Africa.

Pternistes swainsoni (bare-throated francolin): *H.*, Plimmer, 1913, South Africa.

(Ptilinopus iozonus) = **Chlorotreron iozona** (fruit pigeon): *H.*, Plimmer, 1915, Aru Isles.

Ptilonorhynchus violaceus (satin weaver bird): *H.*, and *L.*, Z.S., 1925, New South Wales.

Ptilopachys fuscus (partridge): *H.*, Todd and Wolbach, 1912, Gambia.

Ptilopodiscus coronulatus (lilac-crowned fruit pigeon): *P.* or *H.*, Z.S., 1925, Aru Isles,

(Ptilopus superbus) = **Lamprotreron superba** (fruit pigeon): *H. columbæ*, Breinl, 1913, West Australia.

Ptilotis auricomis (honey-sucker): *H.*, Plimmer, 1915, New South Wales.

Ptilotis chrysops (honey-sucker): *H. ptilotis*, Cleland and Johnston, 1909, Australia.

Ptilotis fusca (honey-sucker): *T. anelobiæ*, Cleland and Johnston, 1911, Australia. *H.*, Cleland and Johnston, 1911, Australia; Plimmer, 1912, Australia. *L. anelobiæ*, Cleland and Johnston, 1911, Australia.

Ptilotis plumula (honey-sucker): *H.*, Johnston, 1910, and Cleland and Johnston, 1910, Australia.

Ptilotis sonora (honey-sucker): *H.*, Cleland and Johnston, 1911, Australia.

Pycnonotus barbatus (bulbul): *H.* and *L.*, Leger, A. and M., 1914, Senegal.

Pycnonotus hainanus = (Ixus hainanus).

(Pycnonotus jocosus) = Otocompsa emeria (bulbul): *P.*, Plimmer, 1912, India.

Pycnonotus tricolor (bulbul): *T. pycnonoti*, Kerandel, 1912, French Congo.

Pycnonotus sp. (bulbul): *T.* and *L.*, Maya and David, 1912, Mauritius.

Pyromelana flammiceps (weaver finch) : *H.* and *L.*, Leger, A. and M., 1914, Senegal.

Pyromelana franciscana (weaver finch): *H.*, Commes, 1918, Niger.

Pyromelana orix = (Euplectes orix).

Pyrrherodias purpurea = (Ardea purpurea).

Pyrrhula sp. (bullfinch): *H.*, Sergent, Ed. and Et., 1904, Algeria.

Quelea erythrops (weaver finch): *H. queleæ* and *P.*, Marullaz, 1912, Africa.

Querquedula circia (garganey): *Hg.*, Franchini, 1924, Italy.

(Querquedula crecca) = Nettion crecca (garganey teal): *L. simondi*, Mathis and Leger, 1910, Tonkin. *Hg.* (?), Franchini, 1923, Italy.

Querquedula querquedula = (Anas querquedula).

Rallus aquaticus (water rail): *H.*, Franchini,1924, Italy.

Ramphastos toco (toucan): *H.*, Iturbe and Gonzalez, 1916, Venezuela.

(Regulus cristatus) = Regulus regulus (golden-crested wren): *T.*, Bettencourt and França, 1907, Portugal.

Regulus ignicapillus (fire-crested wren): *T.*, Bettencourt and França, 1907, Portugal.

Regulus regulus = (Regulus cristatus).

Rhamphocœlus brasilius (tanager): *Hg. (Tx.) rhamphocœli*, Aragão, 1911, Brazil. *P.*, Z.S., 1925, Brazil.

Rhea americana (rhea): *P.*, Carini and Maciel, 1916, Paraguay.

Rhodophoneus cruentus = (Laniarius cruentus).

Rostratula semicollaris = (Tringa atricapilla).

Rupornis leucorrhoa (hawk): *H.*, Iturbe and Gonzalez, 1916, Venezuela.

(Ruticilla aurorea) = Phœnicurus auroreus (redstart): *L.*, Ogawa, 1911, Japan.

(Ruticilla phœnicurus) = Phœnicurus phœnicurus (common redstart): *H.*, Galli-Valerio, 1902, Europe.

(Ruticilla titys) = Phœnicurus titys (black redstart): *T.*, Bettencourt and França, 1907, Portugal.

Sarcidiornis melanonota (wattle duck): *H.*, Rodhain, Pons, Vandenbranden and Bequaert, 1913, Belgian Congo.

Saxicola œnanthe (wheatear): *T.*, Nieschulz, 1921, 1922, Heligoland. *H.*, Galli-Valerio, 1902, Europe; Cardamatis, 1909, Greece.

Saxicola rufa = (Saxicola stapazina).

Saxicola stambajina (? Stapazina = Saxicola rufa) (wheatear): *H.*, Cardamatis, 1909, Greece.

Scardafella squamosa (dove): *P.*, Plimmer, 1912, South America.

Schistochlamys capistrata (tanager): *T. schistochlamydis*, Splendore, 1910, Brazil.

(Scolecophagus carolinus) = Euphagus carolinus (hang-nest): *T. confusum*, Lühe, 1906 (*T. avium*, Novy and McNeal, 1905, North America).

Scolopax rusticola (woodcock): *T.*, Bettencourt and França, 1907, Portugal. *P.*, Z.S., 1925, Europe. *H.*, Ogawa, 1911, Japan; Böing, 1925, Germany; Z.S., 1925, Europe. *L.*, Mathis and Leger, 1911, Tonkin; Ogawa, 1911, Japan. *L. legeri*, França, 1912, Portugal.

Scops bakkamæna (scops owl): *H.*, Castellani and Willey, 1904, Ceylon.

Scops brasiliana (scops owl): *H.*, Leger, 1918, Guiana. *L. lutzi*, Carini, 1920, Brazil. *I.*, *H.*, and *L.*, Lutz and Meyer (quoted by Pinto, 1925), Brazil.

Scops capensis (scops owl): *T.*, *H.*, and *L.*, Rodhain, Pons, Vandenbranden and Bequaert, 1913, Belgian Congo.

(Scops giu) =Scops scops (scops owl): *H.*, Sergent, Ed. and Et., 1905, Algeria.　*L.*, Sergent, Ed. and Et., 1902, Algeria; Plimmer, 1913, 1917, Europe.

Scops leucotis (scops owl): *H.*, Plimmer, 1912, and Z.S., 1925, Gambia.

Scops scops =(Scops giu =Pistrornia scops).

Scops semitorques (scops owl): *T.*, *H.*, and *L.*, Ogawa, 1911, Japan.

Scotopelia bouvieri (owl): *H.*, Plimmer, 1912, South America.

Scotopelia peli (owl): *H.* and *L.*, Rodhain, Pons, Vandenbranden and Bequaert, 1913, Belgian Congo.

Sericulus melinus) =Sericulus chrysocephalus (Regent bird): *P.*, and *H.*, Z.S., 1925, Australia.

Serinus canarius (wild canary): *L.*, Z.S., 1925, Canary Isles.

Serinus icterus =(Crithagra chrysopyga).

Serpentarius serpentarius =(Gypogeranus serpentarius).

Sialia sialis (blue bird, thrush): *T. confusum*, Lühe, 1906 (*T. avium*, Novy and McNeal, 1905, North America); Plimmer, 1913, North America.　*P.* and *H.*, Z.S., 1925, North America.

Sicalis flaveola: *Hg.* (*Tx.*) *sicalidis*, Aragão, 1911, Brazil.

Sitagra intermedius =(Hyphantornis tæniopterus).

Sitagra luteola= (Hypantornis personata).

Sitagra melanocephala =(Hyphantornis melanocephala).

Sitagra monacha (weaver finch): *L.*, Rodhain, 1913, Belgian Congo.

Sitta cæsia (nuthatch): *H.*, Galli-Valerio, 1902, Europe; Cardamatis, 1909, Greece.

Specotheres maxillaris (Australian oriole): *L. anellobiæ*, Cleland and Johnston, 1911, Australia.

Spermophilus sp.: *H.*, Lutz and Meyer (quoted by Pinto, 1925), Brazil.

Spinus citrinellus =(Fringilla citrinella).

Spinus cucullatus =(Chrysomitris cucullatus).

Spinus spinus = (Chrysomitris spinus).

Spizaëtus coronatus (hawk eagle): *H.*, Rodhain, Pons, Vandenbranden and Bequaert, 1913, Belgian Congo.

Sporæginthus melpodus =(Estrilda melpoda) : *P.*, Z.S., 1925, West Africa.

Sporophila albigularis (finch): *Hg.* (*Tx.*) *sporophilæ*, Aragão, 1911, Brazil.

Spreo superbus (superb spreo): *P. præcox* and *H.*, Z.S., 1925, North-East Africa.

Steganopleura guttata (weaver finch): *T.*, Plimmer, 1914, Australia.

Steganura paradisea =(Vidua paradisea).

Stoparola melanops (fly-catcher): *P.*, Plimmer, 1912, Australia.

Streptopelia risoria =(Turtur risorius).

Streptopelia semitorquata =(Turtur semitorquata) (dove): *H.*, Z.S., 1925, Gold Coast.

Streptopelia vinacea (dove): *T.* and *H.*, Todd and Wolbach, 1912, Gambia.

Strix flammea (barn owl): *T.*, Bettencourt and França, 1907, Portugal.　*H. noctuæ*, Celli and San Felici, 1891; Italy.　*H.*, Leger, A. and M., 1914, Niger; Plimmer, 1912, South Africa; Balfour, 1906, Sudan; Sergent, Ed. and Et., 1907, Algeria. *P.*, Sergent, Ed. and Et., 1906, Algeria, Lutz and Meyer (quoted by Pinto, 1925), Brazil.

Strix flammea trimaculata (barn owl): *T.*, Kérandel, 1909, French Congo.　*H.*, Kérandel, 1909 and 1913, French Congo.　*L.*, Kérandel, 1909, French Congo.

(Strix otus) =Asio otus (long-eared owl): *P.*, Wasielewski, 1902 and 1908, Germany. *H.* and *L.*, Wasielewski, 1908, Germany.

Strix perlata =(Tyto perlata).

Strix sp. (barn owl): *L.*, Danilewsky, 1884, Russia.

Struthio camelus (ostrich): *L. struthionis*, Walker, 1912, Africa.

(Sturnella defilippi) =Trupialis defilippi (hang-nest): *H.*, Plimmer, 1913, Chili.

Sturnus menzbieri (common Indian starling): *P. præcox*, Z.S., 1925, India.

Sturnus vulgaris (starling): *H.*, Coles, 1914, England; Celli and San Felici, 1891, Italy; Wasielewski, 1896, Germany; Labbé, 1894, France (?). *L.*, Coles, 1914, England.

Sutoria sutoria = (Orthotomus sutorius).

Sycalis arvensis (misto yellow finch): *L.*, Z.S., 1925, Chili.

Sycalis colombiana (finch): *H.*, Iturbe and Gonzalez, 1916, Venezuela.

Sycalis flaveola (finch): *Hg.* (*Tx.*) *sicalidis*, Aragão, 1911, Brazil. *H.*, Iturbe and Gonzalez, 1916, Venezuela.

Sylvia atricapilla (blackcap warbler): *T.*, Bettencourt and França, 1907, Portugal; Sergent, 1904, Algeria. *H.*, Sergent, Ed. and Et., 1904, Algeria; Wasielewski, 1908, Germany. *P.*, Sergent, Ed. and Et., 1904, Algeria.

(Sylvia cinerea) = Sylvia sylvia (whitethroat): *H.*, Galli-Valerio, 1902, Europe; Cardamatis, 1909, Greece.

Sylvia curruca (lesser whitethroat): *L.*, Z.S., 1925, Europe.

Sylvia hortensis (garden warbler): *H.*, Sjöbring, 1892, Sweden; Wasielewski, 1908, Germany.

(Sylvia hypolais) = Hypolais icterina (icterine warbler): *H.*, Sjöbring, 1892, Sweden.

(Sylvia rubecula) = Erithacus rubecula (robin): *H.*, Cardamatis, 1909, Greece.

(Sylvia sibilatrix) = Phylloscopus sibilator (wood warbler): *H.*, Sjöbring, 1892, Sweden.

Sylvia simplex (warbler): *T.*, Nieschulz, 1921, 1922, Heligoland.

Sylvia sylvia = (Sylvia cinerea).

(Sylvia trochila) = Phylloscopus trochilus (willow warbler): *H.*, Sjöbring, 1892, Sweden.

Sylvia sp. (warbler): *T.* and *H.*, Sergent, Ed. and Et., 1904, Algeria.

Synallaxis ruficapilla (wood-hewer): *P.*, Carini and Maciel, 1916, Paraguay.

Syrnium aluco (tawny owl): *T. avium*, Danilewsky, 1885, Europe; Laveran, 1903, Mayer, 1910. *T. syrnii*, Nöller, 1917, Europe; Plimmer, 1914, Europe. *H. syrnii*, Mayer, 1910, Europe; Plimmer, 1914, Europe. *H.*, Sergent, Ed. and Et., 1905, Algeria; Celli and San Felici, 1891, Italy; Ziemann, 1898, Germany; Danilewsky, 1889, South Russia. *L. danilewskyi*, Ziemann, 1898, Europe; Danilewsky, 1884, Europe; Plimmer, 1914, 1915, Europe; Sergent, 1907, Algeria. *P.*, Sergent, Ed. and Et., 1906, Algeria.

Tachyphonus ornata (? coronata) (tanager): *T.*, De Cerqueira, 1906, Brazil.

(Tadorna tadornoides) = Casarca tadornoides (sheld-duck): *H.*, Plimmer, 1912, Australia.

Tænioptera nengeta (tyrant bird): *H.*, Carini and Maciel, 1916, Paraguay.

Tanagra cana = (Thraupis cana).

Tanagra episcopus (tanager): *H.*, Plimmer, 1912, South America.

Tanagra palmarum (tanager): *H.*, Plimmer, 1912, South America; Iturbe and Gonzalez, 1916, Venezuela. *Hg.* (*Tx.*) *tanagræ*, Aragão, 1911, Brazil.

Tanagra sayaca (tanager): *H.*, Carini and Maciel, 1916, Paraguay.

Tantalus americanus (stork): *T.*, Migone, 1916, Paraguay.

Tetrao urogallus (capercaillie): *L. mansoni*, Sambon, 1908; *T.*, *H.*, *L.*, and *P.*, Böing, 1925, Germany.

Tetrax tetrax = (Otis tetrax).

Textor albirostris = (Textor alecator).

(Textor alecator) = Textor albirostris (weaver finch): *P.*, Plimmer, 1912, West Africa.

Tharrhaleus jerdoni (accentor thrush): *H.*, Plimmer, 1913, India.

Theristicus caudatus (ibis): *T.*, Migone, 1916, Paraguay.

Thrasaëtus harpyia (harpy): *T.*, Iturbe and Gonzalez, 1916, Venezuela.

(Thraupis cana) = Tanagra cana (tanager): *H.*, Iturbe and Gonzalez, 1916, Venezuela.

Thrush sp (?): *L. debreuili*, Mathis and Leger, 1911, Tonkin.

Tigrisoma marmoratum (heron): *T.*, Migone, 1916, Paraguay.

Tinamus subcristatus (tinamu): *T. tinami*, Mesnil, 1912; Brimont, 1912, Guiana.

(Tinnunculus alaudarius) =Cerchneis tinnunculus (kestrel): *H.*, Plimmer, 1912 and 1914, Europe; Danilewsky, 1889, South Russia.

(Tinnunculus cenchris) =Cerchneis naumanni (lesser kestrel): *H.*, Plimmer, 1914, South Europe.

(Tinnunculus tinnunculus) =Cerchneis tinnunculus (common kestrel): *H.*, Ziemann, 1898, Europe.

Tisa variabilis =(Emberiza variabilis).

Toxostoma cinereum (mocking bird): *T.* and *P.*, Plimmer, 1913, California.

Toxostoma rufum =(Harporhynchus rufus).

Tragopan satyra (crimson-horned pheasant): *P. præcox*, Z.S., 1925, Himalayas.

(Treron calva) =Vinago calva (fruit pigeon): *T.*, Wellman, 1905, Angola.

Trichoglossus nigrigularis (lory): *H.*, Plimmer, 1912, New Guinea.

Trichoglossus nigrogenis (lory): *H.*, Plimmer, 1916, Tasmania.

(Tringa atricapilla) =Rostratula semicollaris (painted snipe): *H.*, Leger, 1918, Guiana.

Troglodytes ædon (wren): *T.*, Novy and McNeal, 1905, North America.

(Troglodytes parvulus) =Anorthura troglodytes (wren): *T.*, Novy and McNeal, 1905, North America. *H.*, Galli-Valerio, 1902, Europe.

Trogon atricollis (trogon): *H.*, Iturbe and Gonzalez, 1916, Venezuela.

Tropidorhynchus corniculatus =(Philemon corniculatus) (honey-sucker): *H.*, Cleland and Johnston, 1909, 1911, Australia. *L.*, Breinl, 1911, West Australia.

Trupialis defilippi =(Sturnella defilippi).

Trypanocorax frugilegus =(Corvus frugilegus) (rook): *L.*, Sakharoff, 1893, Trans-caucasia.

Turacus corythraix (turaco): *H.*, Plimmer, 1912, South Africa. *P.*, Plimmer, 1914, South Africa.

Turacus macrorhynchus (turaco): *H.*, Plimmer, 1912, West Africa.

Turacus persa (turaco): *H.*, Plimmer, 1912, West Africa.

Turdus dubius =(Turdus fuscatus).

(Turdus fumigatus) =Merula fumigata (thrush): *H.*, Iturbe and Gonzalez, 1916, Venezuela.

(Turdus fuscatus) =Turdus dubius (thrush): *H.* and *L.*, Ogawa, 1911, Japan.

Turdus migratorius =(Merula migratoria) (American robin, thrush): *P.*, Plimmer, 1916, North America.

(Turdus musicus) =Hylocichla musica (song-thrush): *T.*, Petrie, 1905, England; Coles, 1914, England. *H.*, Coles, 1914, England; Danilewsky, 1889, South Russia; Wasielewski, 1908, Germany. *L.*, França, 1912, Portugal; Coles, 1914, England; Wülker, 1919, Macedonia. *P.*, Coles, 1914, England.

(Turdus mustelinus) =Hylocichla mustelinus (thrush): *P.*, Plimmer, 1914, North America.

Turdus obscurus (thrush): *H.*, Ogawa, 1911, Japan. *L.*, Ogawa, 1912, Japan.

Turdus pallidus (thrush): *H.* and *L.*, Ogawa, 1911, Japan.

(Turdus philomelos) =Hylocichla musica (song-thrush): *T.*, Nieschulz, 1921, 1922, Heligoland.

(Turdus rufiventris) =Merula rufiventer (thrush): *T.* and *Hg.* (*Tx.*), Carini and Maciel, 1916, Brazil.

Turdus torquatus (ring ouzel thrush): *T.*, Nieschulz, 1921, 1922, Heligoland.

(Turtur auritus) =Turtur turtur (dove): *H.*, Sergent, Ed. and Et., 1904, Algeria. *L.*, Leger, 1913, Corsica. *P.*, Sergent, Ed. and Et., 1904, Algeria.

(Turtur humilis) =Onopopelia humilis (dove): *H.* and *L. marchouxi*, Mathis and Leger, 1910, Tonkin.

Turtur orientalis =(Turtur rupicola) (dove): *L.*, Ogawa, 1911, Japan. *P.*, Ogawa, 1912, Japan.

(Turtur risorius) =Streptopelia risoria (dove): *H.*, Cardamatis, 1909, Greece.

(Turtur rupicola) =Turtur orientalis (dove): *T.*, Mathis and Leger, 1911, Tonkin.

(Turtur semitorquata) =Streptopelia semitorquata (dove): *H.*, Rodhain, Pons, Vandenbranden and Bequaert, 1913, Belgian Congo. *L.*, Minchin, 1910, Uganda.

Turtur senegalensis (dove): *H.*, Todd and Wolbach, 1912, Gambia. *L.*, Leger, A. and M., 1914, Senegal.

Turtur turtur =(Turtur auritus) (dove): *H.*, Ziemann, 1898, Europe; Franchini, 1924, Italy; Böing, 1925, Germany. *L.* Franchini, 1924, Italy.

(Tympanistra bicolor) =Tympanistra tympanistra (dove): *P.*, Plimmer, 1912, West Africa.

Tympanistra tympanistra = (Tympanistra bicolor).

Tyrannus melancholicus (tyrant bird): *T.*, Carini and Maciel, 1916, Brazil.

(Tyto perlata) =Strix perlata (owl): *H.*, Iturbe and Gonzalez, 1916, Venezuela.

Upupa epops (hoopoe): *T.*, Bettencourt and França, 1907, Portugal. *H.*, Danilewsky, 1889, South Russia. *L.*, Wülker, 1919, Macedonia.

Uræginthus phœnicotis = (Estrilda phœnicotis).

Uranornis rubra = (Paradisea rubra).

(Urubitinga albicollis) =Leucopternis albicollis (hawk): *H.*, Brimont, 1909, Guiana.

(Urobrachya albonotata) =Penthetria albonotata (weaver bird): *T.*, Plimmer, 1913, East Africa.

Vanellus vanellus (lapwing): *H.* and *L.*, Böing, 1925, Germany.

(Vidua paradisea) =Steganura paradisea (weaver bird): *H.*, Plimmer, 1912 and 1917, West Africa.

(Vidua principalis) =Vidua serena (weaver finch): *T.*, Leger, A. and M., 1914, Niger.

Vidua serena =(Vidua principalis) (weaver finch): *T. viduœ*, Kérandel, 1909, 1912, French Congo.

Vinago calva = (Treron calva).

Vinago nudirostris (fruit pigeon): *H.*, Todd and Wolbach, 1912, Gambia.

Vireo chivi (greenlet): *H.*, Carini and Maciel, 1916, Paraguay.

Volatinia jacarini (finch): *Hg. (Tx.)* sp., Carini and Maciel, 1916, Brazil.

Vultur sp. (vulture): *L.*, Chingareva (Schingareff), 1911, Russia.

(Xiphocolaptes procerus) =Xiphocolaptes promeropirhynchus (wood-hewer). *H.* Iturbe and Gonzalez, 1916, Venezuela.

Xiphocolaptes promeropirhynchus =(Xiphocolaptes procerus).

Zamelodia ludoviciana =(Hedymeles ludovicianus).

Zenaidura carolinensis =(Zenaidura macrura) (dove): *H. maccallumi* and *H. sacharovi*, Novy and McNeal, 1904, North America.

(Zenaidura macrura) =Zenaidura carolinensis (dove): *T.*, Novy and McNeal, 1905, North America.

Zonotrichia gambelii (Gambel's sparrow): *P. præcox*, Z.S., 1925, North America.

(Zonotrichia pileata) =Brachyspiza pileata (finch): *T. zonotrichœ*, Splendore, 1910, South America. *H.*, and *P.*, Lutz and Meyer (quoted by Pinto, 1925), Brazil.

(Zosterops cærulescens) =Zosterops lateralis (white-eye): *H.*, Johnston, 1910, and Cleland and Johnston, 1910, Australia.

Zosterops chloronota = (Zosterops chlorophæa).

(Zosterops chlorophæa) =Zosterops chloronota (white-eye): *T.* and *L.*, Maya and David, 1912, Mauritius.

Zosterops japonica (white-eye): *H.* and *L.*, Ogawa, 1911, Japan.

Zosterops lateralis = (Zosterops cærulescens).

Zosterops simplex (white-eye): *H.*, Mathis and Leger, 1910, Tonkin.

LACERTILIA.

Acanthodactylus boskianus : *Hg. boskiana*, Catouillard, 1909, Tunis.

Acanthodactylus pardalis : *Hg. capensis*, Conor, 1909, Tunis.

Acanthodactylus vulgaris : *Schellackia bolivari*, Reichenow, 1919, Madrid. *Schellackia minuta*, Reichenow, 1919, Madrid.

Acanthosaura fruhstorferi : *T.*, Mathis and Leger, 1911, Tonkin.

Agama colonorum : *T.*, Todd and Wolbach, 1912, Gambia; Macfie, 1914, Nigeria. *Hg. agamæ*, Laveran and Pettit, 1909, Senegal. *Hg. sp.*, Macfie, 1914, Nigeria. *P. agamæ*, Wenyon, 1909, Sudan; Macfie, 1914, Nigeria.

Agama nupta : *H. grahami*, Shortt, 1922, Persia. *Hg. periomsi*, Shortt, 1922, Persia.

Agama tuberculata : *Hg. sp.*, Shortt, 1917, India. *Hg. thomsoni*, Minchin, 1908, India.

Algiroides nigropunctatus : *Hg. sp.*, Plimmer, 1912, Europe.

Ameiva surinamensis : *Hg. ameivæ*, Carini and Rudolph, 1912, Brazil.

Anisolepis undulatus : *Hg. anisolepis*, Marques, 1911, Brazil.

Anolis biporcatus : *H. gonzalezi*, Iturbe and Gonzalez, 1921, Venezuela.

Anolis sp. : *Leishmania henrici*, Leger, 1919, Martinique; Wenyon, 1921.

Callopistes flavipunctatus : *Hg.*, Z.S., 1925, Peru.

Chalcides bedriagæ = (Gongylus ocellatus).

Chamæleon fischeri : *Lankesterella amania*, Awerinzew, 1914, West Africa.

Chamæleon pumilus : *Leishmania chamæleonis*, Bayon, 1914, Robben Island.

Chamæleon senegalensis : *Hg. sp.*, Plimmer, 1912, West Africa.

Chamæleon vulgaris : *T. chamæleonis*, Wenyon, 1909, Sudan. *Leishmania chamæleonis*, Wenyon, 1921, Egypt.

Ctenosaura acanthura = (Cyclura acanthura).

(Cyclura acanthura) = Ctenosaura acanthura : *Hg. sp.*, Plimmer, 1912, Central America.

Diploglossus fasciatus : *P. diploglossi*, Aragão and Neiva, 1909, Brazil.

Gecko verticillatus = (Platydactylus guttatus).

Gerrhosaurus nigrolineatus : *Hg. sp.*, Plimmer, 1913, South Africa.

(Gongylus ocellatus) = Chalcides bedriagæ : *Hg. sergentium*, Nicolle, 1904, Tunis; Viquier and Weber, 1912, Africa.

Hemidactylus gleadovii : *T. hemidactyli*, Mackie, Gupta and Swaminath, 1923, India. *Leishmania hemidactyli*, Mackie, Gupta and Swaminath, 1923, India.

Hemidactylus leschenaultii : *T. leschenaulti*, Robertson, 1908, Ceylon. *T. pertenue*, Robertson, 1908, Ceylon. *H. simondi*, Castellani and Willey, 1904, Ceylon; Robertson, 1908, Ceylon; Dobell, 1910, Ceylon.

Hemidactylus triedrus : *T. pertenue*, Robertson, 1908, Ceylon. *Hg. tiedri*, Robertson, 1908, Ceylon.

Hemidactylus sp. : *H.*, Donovan (first record), India.

Iguana delicatissima = (Iguana nudicollis).

(Iguana nudicollis) = Iguana delicatissima : *P. carinii*, Leger and Mouzels, 1917, South America.

(Iguana sapidissima) = Iguana tuberculata : *P. minasense*, Wenyon, 1915, Trinidad.

Iguana tuberculata = (Iguana sapidissima) : *Hg. iguanæ*, Laveran and Nattan-Larrier, 1912, South America; Darling, 1912, Panama; Plimmer, 1912, 1913, and Z.S., 1925, South America.

Lacerta agilis : *Hg. lacertarum*, Danilewsky, 1886, Europe. *Hg. lacazei*, Labbé, 1894, Europe.

Lacerta galloti : *Hg. sp.*, Plimmer, 1914, North Africa; Plimmer, 1912, Canary Isles.

Lacerta muralis : *Hg. berestnewi*, Finkelstein, 1908, Russia. *Hg.* sp., Laveran and Pettit, 1908, France; Z.S., 1925, South Europe. *Hg. lacertarum*, Danilewski, 1886, Europe. *Hg. lacazei*, Labbé, 1894, Europe. *Hg. bicapsulata*, França, 1909, Portugal. *Hg. marceaui*, França, 1909, Portugal. *Hg. nana*, França, 1909, Portugal. *Hg. nobrei*, França, 1909, Portugal. *Karyolysus lacertæ*, Labbé, 1894, Europe; Reichenow, 1913, 1921, Europe. *Karyolysus bicapsulatus*, Reichenow, 1921, Europe. *Karyolysus lacazei*, Reichenow, 1913, 1921, Europe; Woodcock, 1912, Italy. *Eutrichomastix lacertæ*, Reichenow, 1918, 1920, Spain.

Lacerta ocellata : *Schellackia minuta*, Reichenow, 1921, Madrid. *Hg. lacertarum*, Danilewsky, 1886, Europe. *Hg. curvirostris*, Billet, 1904, Algiers; França, 1909, Portugal. *Hg. biretorta*, Nicolle, 1904, Tunis; França, 1909, Portugal. *Hg. schaudinni*, França, 1908, Portugal. *Hg. schaudinni* var. *africana*. França, 1908, Portugal. *Hg. minuta*, França, 1909, Portugal. *Hg. nicollei*, França, 1909, Portugal. *Hg.* sp., Plimmer, 1912, 1913, 1914, 1917, Europe.

Lacerta ocellata var. pater : *Hg. curvirostris*, Laveran and Pettit, 1909, Tunis; Manceaux, 1912, Tunis. *Hg. biretorti*, Manceaux, 1912, Tunis. *Hg. schaudinni*, Manceaux, 1912, Tunis.

Lacerta peloponnesiaca : *Hg.* sp., Plimmer, 1912, Europe.

Lacerta viridis : *Hg. lacertarum*, Danilewsky, 1886, Europe; Laveran and Pettit, 1908, France. *Karyolysus biretortus*, Reichenow, 1921, Madrid.

Lygosoma sp.: *T.*, Todd and Wolbach, 1912, Gambia.

Lygosoma quoyi : *Hg. hinuliæ*, Johnston and Cleland, 1910, Australia.

Lygosoma tæniolatum : *T.* and *Hg.*, Johnston and Cleland, 1912, Australia.

Mabuia agilis : *T.*, Carini, 1912, Brazil. *T. rudolphi*, Carini, 1913, Brazil. *P. minasense*, Carini and Rudolph, 1912, Brazil.

Mabuia maculilabris : *T. martini*, Bouet, 1909, Ivory Coast.

Mabuia perrotetii : *T. martini*, Bouet, 1909, Ivory Coast. *T. perroteti*, França, 1911, Portuguese Guinea.

Mabuia quinquetæniata : *T. mabuiæ*, Wenyon, 1909, Sudan. *Hg. gracilis*, Wenyon, 1909, Sudan. *P. mabuiæ*, Wenyon, 1909, Sudan.

Mabuia raddonii : *T. boueti*, Martin, 1907, French Guinea; Leger, M. and A., 1914, Senegal.

Mabuia varia : *Hg.*, Fantham, 1919, South Africa.

Mabuia vittata : *Hg. mabuiæ*, Nicolle and Comte, 1906, North Africa.

Macroscincus coctæi : *Hg. macroscinci*, Laveran, 1907, Cape Verde.

Ophisaurus apus : *Hg.* sp., Finkelstein, 1908, Russia; Plimmer, 1912, Europe.

Ophisaurus ventralis : *Hg.*, Z.S., 1925, North America.

Phyllodactylus elisæ : *Hg. phyllodactyli*, Shortt, 1922, Persia. *Hg. procteri*, Shortt, 1922, Persia.

Phyllodactylus gerrhopygus : *Hg.* sp., Escomel, 1917, Peru.

(Platydactylus guttatus) = Gecko verticillatus : *Hg.* sp., Prowazek, 1907, Japan.

(Platydactylus mauritanicus) = Tarentola mauritanica : *Hg. platydactyli*, Billet, 1904, Algiers.

(Platydactylus muralis) = Tarentola mauritanica : *T. platydactyli*, Catouillard, 1909, Tunis.

Psammodromus algirus : *Hg. psammodromi*, Soulié, 1904, Algiers; França, 1909, Portugal. *Hg. lusitanica*, França, 1909, Portugal. *Hg. pallida*, França, 1908, Portugal.

Psammodromus hispanicus : *Schellackia bolivari*, Reichenow, 1919, Madrid. *Schellackia minuta*, Reichenow, 1921, Madrid.

Psilodactylus caudicinctus : *T. gallayi*, Bouet, 1909, Africa.

Ptyodactylus lobatus : *Hg.* sp., Plimmer, 1912, Egypt.

(Sceloporus clarkii) = Sceloporus spinosus var. clarkii : *Hg* sp., Plimmer, 1912, North America.

Sceloporus spinosus var. clarkii = (Sceloporus clarkii).

Tarentola annularis : *Hg.* sp., Plimmer, 1912, Egypt.

Tarentola mauritanica =(Platydactylus mauritanicus =Platydactylus muralis) : *T. platydactyli,* Chatton and Blanc, 1915, North Africa; Plimmer, 1912, North Africa. *Pirhemocyton tarantolæ,* Chatton and Blanc, 1914, Tunis. *Leishmania tarantolæ,* Chatton and Blanc, 1918, North Africa; Wenyon, 1921. *Trichomastix,* Chatton, 1918, North Africa. *Hg. platydactyli,* Foley and Catanei, 1926, Algeria.

Tiliqua scincoides : *Hg. tiliquæ,* Johnston and Cleland, 1912, Australia.

Tropidurus torquatus : *P. tropiduri,* Aragão and Neiva, 1909, Brazil. *Hg.* sp., Phisalix and Tejera, 1920, Venezuela.

Tupinambis nigropunctatus : *Hg.* sp., Leger and Mouzels, 1917, South America.

Tupinambis teguixin : *Hg. tupinambis,* Laveran and Salimbeni, 1909, Brazil; Carini, 1909, Brazil. *Hg. missoni,* Carini, 1909, Brazil. *Hg. iguanæ,* Ducceschi, 1914, Brazil. *Hg.* sp., Plimmer, 1912, 1913, South America.

(Varanus arenarius) =Varanus griseus : *Hg.* sp., Marchoux, 1908, Senegal; Bouet, 1909, West Africa.

Varanus bengalensis =(Varanus dracæna) : *Hg.* sp., Plimmer, 1912, 1914, India.

(Varanus bivittatus) =Varanus salvator : *Hg.* sp., Prowazek, 1912, Sumatra.

(Varanus dracæna) =Varanus bengalensis : *Hg.* sp., Simond, 1901, India.

Varanus exanthematicus : *T. varani,* Lloyd, Johnson, Young and Morrison, 1924, Nigeria.

Varanus gouldii : *Hg. gouldii,* Johnston and Cleland, 1912, Australia.

Varanus griseus =(Varanus arenarius) : *Hg. borreli,* Nicolle and Comte, 1906, Tunis; Foley and Catanei, 1926, Algeria.

Varanus nebulosa : *Hg.,* Z.S., 1925, Malaya.

Varanus niloticus : *T. varani,* Wenyon, 1909, Sudan; Lloyd, Johnson, Young and Morrison, 1924, Nigeria. *Hg. varani,* Laveran, 1905, Transvaal; França, 1910, Portuguese Guinea. *Hg. borreli,* Nicolle and Comte, 1906, Tunis. *Hg. toddi,* Wolbach, 1914, Gambia. *Hg.* sp., Leger, M. and A., 1914, Senegal; Wenyon, 1909, Sudan; Plimmer, 1912, Africa.

Varanus salvator =(Varanus bivittatus).

Varanus varius : *Hg.* sp., Gilruth, 1910, Australia; Plimmer, 1912, Australia. *Hg. varanicola,* Johnston and Cleland, 1910, Australia.

OPHIDIA.

Ancistrodon contortrix : *Hg.* sp., Langmann, 1899, North America; Plimmer, 1913, Texas.

Ancistrodon piscivorus : *Hg. morcassini,* Laveran, 1902, North America. *Hg.* sp., Langmann, 1899, North America; Plimmer, 1913, and Z.S., 1925, North America.

Atheris chlorechis : *Hg.* sp., Plimmer, 1912, West Africa.

Atractaspis microlepidota : *Hg.* sp., Wenyon, 1909, Sudan.

Bitis arietans : *T.,* Dutton, Todd and Tobey, 1907, Gambia; Plimmer, 1912, South Africa. *Hg. arietans,* Fantham, 1925, South Africa.

Bitis gabonica : *Hg. dogieli,* Hoare, 1921, Uganda.

Boa constrictor : *Hg. serpentium,* Lutz, 1901, South America. *Hg. leizii,* Sambon, 1907, South America; Marullaz, 1912, North America; Marullaz and Roudsky, 1913, South America. *Hg.* sp., Dobell, 1908, Brazil; Plimmer, 1912, 1913, 1914, 1915, 1916, 1917, and Z.S., 1925, South America.

Boa imperator : *Hg. imperatoris,* Seidelin, 1911, Mexico; Darling, 1912, Panama; Plimmer, 1915, Central America.

Boa madagascariensis : *Hg.* sp., Plimmer, 1912, Madagascar.

Boodon fuliginosus : *Hg. boodoni,* Phisalix, 1914, Africa; Plimmer, 1914, West Africa.

(Bothrops sp.) =Lachesis sp. : *Hg.* sp., Lutz, 1901, South America.

(Bothrops viridis) =Lachesis gramineus : *Hg.* sp., Simond, 1901, Cochin-China.

Bungarus cæruleus = Bungarus candidus : *Hg.* sp., Patton, 1909, India.

Bungarus candidus = Bungarus cæruleus : *Hg.* sp., Patton (Dobell), 1908, India; Plimmer, 1913, India.

Bungarus fasciatus : *Hg. bungari*, Billet, 1895, Tonkin; Mathis and Leger, 1911. Tonkin.

Calabaria reinhardti : *Hg.* sp., Plimmer, 1913, West Africa.

Causus rhombeatus : *Hg.* sp., Bouet, 1909, West Africa; Plimmer, 1914, South Africa.

Cerastes cornutus : *Hg. seurati*, Laveran and Pettit, 1911, and Foley and Catanei, 1925, Algeria.

Chlorophys emini : *Hg.* sp., Wenyon, 1909, Sudan.

Chrysopelea ornata : *Hg.* sp., Robertson, 1908, Ceylon.

Cœlopeltis monopessulana : *Hg.* sp., Z.S., 1925, South Europe.

(Coluber æsculapii) = Coluber longissimus : *Hg. colubri*, Börner, 1901, Europe.

Coluber catenifer : *Hg.* sp., Z.S., 1925, California.

Coluber corais = (Spilotes couperi) : *Hg. rarefasciens*, Sambon, 1907, South America. Lutz, 1901, and Z.S., 1925, South America; Plimmer, 1912, 1915, Brazil.

Coluber guttatus : *Hg.* sp., Plimmer, 1912, North America; Z.S., 1925, south-east of North America.

Coluber helena : *Hg.* sp., Plimmer, 1914, Ceylon.

Coluber lætus : *Hg.* sp., Plimmer, 1916, North America.

Coluber leopardinus : *Hg.* sp., Plimmer, 1914, and Z.S., 1925, Europe. *Trichomonas* sp., Plimmer, 1912, Europe.

Coluber longissimus = (Coluber æsculapii) : *Hg. colubri*, Börner, 1901, North America; Finkelstein, 1908, Caucasia; Plimmer, 1912, 1916, Europe.

Coluber melanoleucus = (Pituophis melanoleucus = Pituophis sayi) : *Hg. brumpti*, Sambon, 1907, Mexico; Plimmer, 1912, 1915, Mexico. *T.x.* sp., Plimmer, 1916, Mexico.

Coluber obsoletus : *Hg.* sp., Plimmer, 1912, 1914, 1915, and Z.S., 1925, North America.

Coluber quatuorlineatus : *Hg.* sp., Dobell, 1908.

Coluber radiatus : *Hg.* sp., Z.S., 1925, Hong-Kong.

Coluber sp. : *Hg.* sp., Simond, 1901, India.

Coluber triaspis : *Hg.* sp., Laveran and Pettit, 1909, Mexico.

Corallus caninus : *Hg.* sp., Brimont, 1909, French Guiana.

Corallus cookii : *Hg.* sp., Langmann, 1901, North America. *Hg. luhei*, Sambon, 1907, America; Plimmer, 1912, 1916, Trinidad.

Coronella getula = (Lampropeltis getulus) : *Hg.* sp., Langmann, 1899, North America. *Hg. wardi*, Sambon, 1907, North America; Plimmer, 1912, 1913, 1914, 1917, and Z.S., 1925, North America.

Coronella girondica : *Hg. coronellæ*, França, 1910, Portugal.

(Crotalus adamanteus) = Crotalus scutulatus : *Hg.* sp., Langmann, 1899, North America.

Crotalus atrox = Crotalus confluentus : *Hg.* sp., Plimmer, 1912, 1913, 1914, 1915, North America.

Crotalus confluentus = Crotalus atrox : *Hg. crotali*, Laveran, 1902, North America; Langmann, 1899; Plimmer, 1912, North America.

Crotalus exsul : *Hg.*, Z.S., 1925, North America.

Crotalus horridus : *Hg.* sp., Plimmer, 1912, North America.

Crotalus scutulatus = (Crotalus adamanteus).

Crotalus oregonus : *Hg.*, Z.S., 1925, North America.

Crotalus sp. : *Hg.* sp., Lutz, 1901, South America.

Dendraspis viridis : *Hg.* sp., Plimmer, 1912, 1913, 1915, West Africa.

Dendrophis pictus : *Hg.* sp., Patton, 1909, India.

Dendrophis punctulatus : *Hg. dendrophidis*, Johnston and Cleland, 1910, Australia.

Diemenia psammophis : *Hg.* sp., Johnston and Cleland, 1910, Australia.

Diemenia textilis : *T.* sp., Love, quoted by Johnston and Cleland, 1910, Australia.

Dipsadomorphus fuscus : *Hg.* sp., Breinl, 1912, Australia.

Drymobius bifossatus : *Hg.* sp., Lutz, 1901, South America. *Hg. drymobii*, Marullaz, 1912, Brazil.

Drymobius boddærtii : *Hg.* sp., Plimmer, 1912, Central America.

Dryophis mycterizans : *Hg.* sp., Patton, 1909, India.

Dryophis prasinus : *Hg.* sp., Z.S., 1925, Hong-Kong and Malaya.

Echis carinatus : *Hg.* sp., Plimmer, 1913, North Africa.

Elaps fulvius : *Hg.* sp., Lutz, 1901, South America; Plimmer, 1914, and Z.S., 1925, North America.

Epicrates cenchris : *Hg.* sp., Darling, 1912, Panama; Plimmer, 1913, and Z.S., 1925, Trinidad.

Erythrolamprus æsculapii : *T. erythrolampri*, Wenyon, 1909, South America. *Hg. colubri*, Börner, 1901, America; Wenyon, 1909, South America.

Eryx conicus = (Gongylophis conicus) : *Hg.* sp., Laveran, 1905, India. *Hg. cantliei*, Sambon, 1907, India.

Eryx jaculus : *Hg.*, Z.S., 1925, North America.

Eryx johnii : *Hg.* sp., Patton, 1909, India; Plimmer, 1912, 1913, and Z.S., 1925, India.

Eryx thebaicus : *Hg.* sp., Plimmer, 1916, 1917, West Africa.

Eunectes murinus : *Hg. serpentium*, Lutz, 1901, South America; Plimmer, 1914, 1916, and Z.S., 1925, South America.

(Eutænia saurita) = Tropidonotus sauritus : *Hg.* sp., Langmann, 1901, North America.

(Eutænia sirtalis) = Tropidonotus ordinatus : *Hg.* sp., Langmann, 1901, North America.

Gastropyxis smaragdina : *Hg.* sp., Plimmer, 1914, West Africa.

(Gongylophis conicus) = Eryx conicus : *Hg.* sp., Patton, 1909, India.

Grayia smythii : *T. clozeli*, Bouet, 1909, West Africa.

Grayia tholloni : *Hg.* sp., Wenyon, 1909, Sudan.

Helicops modestus (water snake): *T. brazili*, Brumpt, 1914, 1915, Brazil.

Herpetodryas carinatus : *Hg.* sp., Lutz, 1901, South America.

Heterodon platyrhinos : *Hg.* sp., Plimmer, 1914, 1915, 1916, North America.

Heterodon simus : *Hg.* sp., Plimmer, 1912, North America. *Trichomonas* sp., Plimmer, 1912, North America.

Hypsirhina chinensis : *T. primeti*, Mathis and Leger, 1909, 1911, Tonkin. *Hg.* sp., Mathis and Leger, 1911, Tonkin.

Lachesis alternatus : *Hg. roulei*, Phisalix and Laveran, 1913, Brazil.

Lachesis gramineus = (Bothrops viridis).

Lachesis lanceolatus : *Hg. plimmeri*, Sambon, 1909, South America; Phisalix, 1912, 1913, South America.

Lachesis mutus : *Hg. seligmanni*, Sambon, 1907, South America; Plimmer, 1914, Trinidad.

Lachesis sp. = (Bothrops sp.).

(Lampropeltis getulus) = Coronella getula : *Hg.* sp., Lutz, 1901, South America.

Leptodira albofusca : *Hg.* sp., Darling, 1912, Panama.

Leptodira annulata : *Hg.* sp., Plimmer, 1917, South America.

(Leptodira attarensis) = Leptodira degeni : *Hg.* sp., Wenyon, 1909, Sudan.

Leptodira degeni = (Leptodira attarensis).

Leptodira hotambœia : *Hg.* sp., Plimmer, 1916, West Africa.

Leptophis liocercus : *Hg.* sp., Plimmer, 1912, 1916, South America.

Leptophis viperinus : *Hg.* sp., Plimmer, 1917, Europe.

Lytorhynchus diadema : *Hg. gigantinensis*, Cuénod, 1909, Tunis.

Macroprotodon cucullatus : *Hg. joannovi*, Hagenmüller, 1898, Algeria and Spain.

(Morelia spilotes) =Python spilotes.

Naja bungarus : *Hg.* sp., Plimmer, 1913, and Z.S., 1925, India.

(Naja hajæ) =Naja tripudians : *Hg. weissi,* Conor, 1912, Africa; Wenyon, 1909, Sudan. *H. najæ,* Wenyon, 1909, Sudan.

Naja nigricollis : *T. najæ,* Wenyon, 1909, Sudan. *T. voltariæ,* Macfie, 1919, Gold Coast. *Hg.* sp., Wenyon, 1909, Sudan; Plimmer, 1916, West Africa. *H. najæ,* Wenyon, 1909, Sudan; Macfie, 1919, Gold Coast.

Naja tripudians =(Naja hajæ) : *Hg. najæ,* Laveran, 1902, India; Patton, 1909, India; Plimmer, 1912, 1913, 1914, 1916, and Z.S., 1925, India. *Trichomonas* sp., Plimmer, 1912, India.

Naja tripudians var. atra : *Hg.* sp., Simond, 1901, India.

Naja sp.: *Hg.* sp., Simond, 1901, India; Bouet, 1909, West Africa.

Natrix tigrina : *Hg. tigrinæ,* Hoare, 1918, Japan.

Philodryas baroni : *Hg.* sp., Senez, 1918, Argentine.

Philodryas olfersii : *Hg.* sp., Lutz, 1901, South America.

Philodryas schotti : *Hg. philodryasi,* Carini, 1910, Brazil; Plimmer, 1913, South America.

Philothamnus semivariegatus : *Hg.* sp., Bouet, 1909, West Africa.

Philothamnus sp.: *Hg.* sp., Bouet, 1909, West Africa.

Phrynonax sulphureus : *Hg.* sp., Plimmer, 1914, Trinidad.

Pituophis catenifer : *Hg.* sp., Z.S., 1925, California.

(Pituophis melanoleucus) =Coluber melanoleucus : *Hg. pituophis,* Laveran and Pettit, 1909, Mexico; Z.S., 1925, North America.

(Pituophis sayi) =Coluber melanoleuchus : *Hg.* sp., Plimmer, 1912, 1913, 1914, North America.

Psammophis elegans : *Hg.* sp., Plimmer, 1916, West Africa.

Psammophis sibilans : *Hg. brendæ,* Sambon, 1907, Africa; Wenyon, 1909, Sudan; Z.S., 1925, Gambia.

Psammophis subtæniatus : *Hg.* sp., Wenyon, 1909, Sudan.

Psammophis trigrammus : *Hg.* sp., Bouet, 1909, West Africa.

Pseudaspis cana : *Hg. refringens,* Sambon, 1907, South Africa; Plimmer, 1912, South Africa.

Pseudechis australis : *Hg. bancrofti,* Johnston and Cleland, 1912, Australia.

Pseudechis mortonensis : *Hg. bancrofti,* Johnston and Cleland, 1912, Australia.

Pseudechis porphyriacus : *Hg. pseudechis,* Johnston, 1909, Australia.

Python amethystinus : *Hg. amethystina,* Johnston, 1909, Australia.

Python molurus : *Hg.* sp., Patton, 1909, India; Phisalix, 1913, India. *Hg. pococki,* Sambon, 1907, India; Plimmer, 1912, 1913, 1914, 1916, 1917, and Z.S., 1925, India.

Python regius : *Hg.* sp., Bouet, 1909, West Africa. *Hg. robertsoni,* Sambon, 1909, West Africa.

Python reticulatus : *Hg. pythonis,* Billet, 1895, Tonkin; Prowazek, 1912, Sumatra; Plimmer, 1912, East Indies.

Python sebæ : *Hg. sebai,* Laveran and Pettit, 1909, Senegal; Bouet, 1909, West Africa; Plimmer, 1912, Africa. *Trichomonas* sp., Plimmer, 1912, West Africa.

Python sp.: *Hg.,* Robertson, 1906, India. *Hg. pythonis,* Billet, 1895, Tonkin; Prawazek, 1908, Java. *Hg.* sp., Wenyon, 1909, Sudan.

Python spilotes =(Morelia spilotes) : *Hg. shattocki,* Sambon, 1907, Australia; Laveran, 1908, Australia; Johnston, 1909, Australia. *Hg.* sp., Laveran, 1908, Australia. *Hg.* sp., Dobell, 1908, Australia; Plimmer, 1912, 1914, Australia. *Hg. megalocystis,* Gilruth, Sweet and Dodd, 1910, Australia.

Python spilotes var. variegata : *Hg. moreliæ,* Johnston, 1910, Australia. *Hg. megalocystis,* Gilruth and Sweet, 1910, Australia.

Rhadinæa merremii : *T.,* Brumpt, 1914, Brazil; *Hg.* sp., Lutz, 1901, South America.

Rhamphiophis rubropunctatus : *Hg. vaughani*, Balfour, 1909, Sudan.

Sepedon hæmachates : *H. mesnili*, Leger, M. and A., 1914, Senegal.

Sepedon sp.: *H. mesnili*, Bouet, 1909, Senegal.

Sistrurus miliarius : *Hg.* sp., Plimmer, 1912, 1914, 1915, North America.

(Spilotes couperi) =Coluber corais : *Hg.* sp., Lutz, 1901, South America.

Spilotes pullatus : *Hg.* sp., Lutz, 1901, South America.

Tarbophis fallax : *Hg.* sp., Plimmer, 1912, 1913, 1914, and Z.S., 1925, Europe.

Tropidonotus asperrimus : *Hg. mirabilis*, Castellani and Willey, 1904, Ceylon.

Tropidonotus fasciatus : *Hg. bradfordi*, Sambon, 1909; Langmann, 1901, North America; Plimmer, 1912, North America. *Amœbæ* (?), Plimmer, 1916, North America.

Tropidonotus ferox : *T. clozeli*, Bouet, 1909, West Africa.

Tropidonotus hammondi : *Hg.*, Z.S., 1925, North America.

Tropidonotus ordinatus =(Eutænia sirtalis).

Tropidonotus piscator : *T. primeti*, Mathis and Leger, 1911, Tonkin. *Hg. mirabilis*, Castellani and Willey, 1904, Ceylon; Plimmer, 1913, and Z.S., 1925, India; Patton, 1909, India.

Tropidonotus sauritus =(Eutænia saurita).

Tropidonotus stolatus : *Hg.* sp., Billet, 1895, Tonkin; Patton, 1909, India.

Tropidonotus taxispilotus : *Hg.* sp., Z.S., 1925, California.

Tropidonotus viperinus : *Hg. viperini*, Billet, 1904, Algeria. *Amœbæ* (?), Plimmer, 1916, 1917, Europe.

Vipera ammodytes (sand viper): *Hg.* sp., Plimmer, 1912, 1913, and Z.S., 1925, Europe.

Vipera aspis : *Hg. samboni*, Giordano, 1907, Europe; Sambon, 1907, Europe.

Vipera libetina : *Hg.* sp., Sergent, 1918, Algeria.

Vipera russellii : *Hg.* sp., Patton, 1909, India; Plimmer, 1912, 1913, 1915, India.

Xenodon neuwiedii : *Hg.* sp., Lutz, 1901, South America.

Zamenis algirus : *Hg. algira*, Manceaux, 1908, Algeria.

Zamenis constrictor : *Hg.* sp., Laveran and Pettit, 1909, Mexico; Plimmer, 1912, 1915, North America.

Zamenis dahlii : *Hg.* sp., Plimmer, 1912, Europe.

Zamenis flagelliformis : *Hg. mansoni*, Sambon, 1907, Central America; Plimmer, 1912, 1914, 1915, North America.

Zamenis gemonensis : *Hg.* sp., Plimmer, 1912, 1913, 1914, 1915, Europe.

Zamenis grahami : *Hg.* sp., Plimmer, 1914, North America.

Zamenis hippocrepis : *Hg. zamensis*, Laveran, 1902, Algeria; Manceaux, 1908, Algeria; Plimmer, 1912, Europe. *Hg. manceauxi*, França, 1910, Tunis. *Hg. luisieri*, França, 1910, Portugal.

Zamenis mucosus : *Hg.* sp., Robertson, 1908, Ceylon; Patton, 1909, India; Plimmer, 1912, 1913, 1914, India.

CHELONIA.

Batagur baska : *Hg.* sp., Plimmer, 1913, Perak.

Chelodina expansa : *Hg.* sp., Plimmer, 1915, Australia.

Chelodina longicollis : *T. chelodina*, Johnson, 1907, Johnston and Cleland, 1912, Australia. *Hg. chelodinæ*, Laveran and Pettit, 1909, Algiers. *Hg. clelandi*, Johnston and Cleland, 1912, Australia. *H. chelodinæ*, Johnston and Cleland, 1909, Australia.

Chelodina oblonga : *Hg. clelandi*, Johnston, 1909, Australia. *H. chelodinæ*, Johnston and Cleland, 1909, Australia.

Chitra indica =(Trionyx indicus).

Chrysemys picta : *Hg.* sp., Plimmer, 1912, North America. *H.* sp., Plimmer, 1912, North America.

Chrysemys scripta var elegans =(Clemmys elegans).

Cinixys belliana : *Hg. sternotheri*, França, 1900, Portuguese Guinea; Plimmer, 1912, Central Africa. *H. roumei*, Bouet, 1909, West Africa; Plimmer, 1912, West Africa.

Cinixys erosa : *H.* sp., Plimmer, 1912, West Africa.

Cinixys homeana : *T. leroyi*, Commes, 1919, Senegal. *Hg.* sp., Commes, 1919, Senegal. *H.* sp., Plimmer, 1912, West Africa.

Cinosternum cruentatum : *Hg.* sp., Plimmer, 1913, 1914, 1916, Central America.

Cinosternum oderatum : *Hg.*, Z.S., 1925, North America.

Cinosternum pennsylvanicum : *Hg.* sp., Plimmer, 1916, North America.

Cistudo carolina : *Octomitus* sp., Plimmer, 1912, 1915, North America.

(Cistudo europæa) =Emys orbicularis : *Hg. stepanowi*, Danilewsky, 1885, Europe.

Clemmys africana : *H. cajali*, Pittaluga, 1912, West Africa.

(Clemmys elegans) =Chrysemys scripta var. elegans: *Hg. labbei*, Börner, 1901, North America.

Clemmys guttata : *Hg.* sp., Plimmer, 1912, North America.

Clemmys japonica : *Hg.* sp., Koidzumi, 1910, Japan. *Hg. clemmydis*, Prowazek, 1910, Japan.

Clemmys leprosa =(Emys leprosa) : *Hg.* sp., França, 1910, Portugal. *Hg. bagensis*, Doucloux, 1904, Tunis; Laveran and Pettit, 1909, Tunis. *Hg.* sp., Plimmer, 1912, Spain.

(Cryptopus granosus) =Emyda granosa : *Hg. laverani*, Simond, 1901, India.

Cyclemys amboinensis : *Hg.* sp., Plimmer, 1913, Malaya. *Trichomonas* sp., Plimmer, 1913, and Z.S., 1925, Malaya.

Cyclemys trifasciata : *Octomitus* sp., Plimmer, 1912, East Indies.

Damonia reevesii : *T. damoniæ*, Laveran and Mesnil, 1902, Ceylon. *Hg. stepanowiana*, Laveran and Mesnil, 1902, Ceylon. *Hg. rara*, Laveran and Mesnil, 1902, Ceylon. *Hg.* sp., Plimmer, 1912, 1914, China.

Damonia subtrijuga : *Hg. pellegrini*, Laveran and Pettit, 1910, Asia.

Emyda granosa =(Cryptopus granosus) : *Hg. nicoriæ*, Patton, 1908, India.

Emyda japonica : *Hg. emydæ*, Prowazek, 1910, Japan.

Emyda vittata : *T. vittatæ*, Robertson, 1908, Ceylon. *Hg. vittatæ*, Robertson, 1908, Ceylon; Donovan (first record), India.

Emydura krefftii : *H. chelodinæ*, Johnston and Cleland, 1910, 1912, Australia. *T. chelodina*, Johnston and Cleland, 1910, 1912, Australia. *Hg. clelandi*, Johnston and Cleland, 1910, 1912, Australia.

Emys blandingii =(Emys meleagris).

(Emys leprosa) =Clemmys leprosa : *Hg. bagensis*, Ducloux, 1904, Algiers.

(Emys meleagris) =Emys blandingii : *Hg.* sp., Hahn, 1909, North America.

Emys orbicularis =(Cistudo europæa) : *Hg.* sp., França, 1910, Portugal; Plimmer, 1912, 1913, and Z.S., 1925, Europe. *Hg. stepanowi*, Danilewsky, 1885, Europe: Reichenow, 1910, Europe.

(Emys tecta) =Kachuga tectum : *Hg. mesnili*, Simond, 1901, India.

Geœmyda spinosa : *Hg.* sp., Plimmer, 1914, Malaya.

Hydraspis hilarii : *Hg.* sp., Plimmer, 1912, Brazil.

Kachuga tectum =(Emys tecta).

Nicoria punctularia : *Octomitus* sp., Plimmer, 1914, South America.

Nicoria trijuga : *Hg. nicoriæ*, Castellani and Willey, 1904, Ceylon; Alexeieff, 1912, Ceylon; Robertson, 1908, Ceylon.

Ocadia sinensis : *Hg.* sp., Koidzumi, 1910, Tonkin; Mathis and Leger, 1911, Tonkin.

Platemys sp.: *Hg. labbei*, Börner, 1901, North America.

Podocnemis expansa : *Hg.* sp., Plimmer, 1912, South America.

Staurotypus triporcatus : *Hg.* sp., Plimmer, 1912, Central America. *H.* sp., Plimmer, 1912, Central America.

Sternothærus adansonii : *Hg.* sp., Wenyon, 1909, Sudan.

Sternothærus derbianus : *T. pontyi*, Bouet, 1909, Africa. *Hg.* sp., Bouet, 1909, Africa. *Hg. sternothœri*, França, 1910, Portuguese Guinea.

Sternothærus niger : *Hg.* sp., Plimmer, 1912, West Africa.

Testudo angulata : *Hg.* sp., Plimmer, 1913, South Africa. *Octomitus* sp., Plimmer, 1912, South Africa.

Testudo emys : *Hg. testudinis*, Laveran and Nattan-Larrier, 1912, India; Plimmer, 1913, and Z.S., 1925, Malaya.

(Testudo ibera) = Testudo mauritanica : *Hg.* sp., Bâznosano, 1901, Roumania; Z.S., 1925, South Europe and Algeria.

Testudo marginata : *Hg.* sp., Pfeiffer, 1891, Greece.

Testudo mauritanica = (Testudo ibera) : *Hg. mauritanica*, Sergent, Ed. and Et., 1904, Algiers.

Testudo pardalis : *H. testudinis*, Laveran, 1905, South Africa.

Testudo tabulata : *Hg. dimorphon*, Brimont, 1909, French Guiana.

Trionyx cartilagineus = (Trionyx stellatus) : *Hg.* sp., Mathis and Leger, 1911, Tonkin.

(Trionyx indicus) = Chitra indica : *H. metchnikowi*, Simond, 1901, India.

(Trionyx niloticus) = Trionyx triunguis : *Hg.*, Stevenson, 1911, Sudan.

(Trionyx stellatus) = Trionyx cartilagineus : *Hg. billeti*, Simond, 1901, Tonkin.

Trionyx triunguis = (Trionyx niloticus) : *Hg. trionyxis*, Thiroux, 1911, Senegal. *Hg.* sp., Plimmer, 1916, West Africa.

Trionyx sp.: *Hg. trionyxis*, Thiroux, 1911, Senegal.

CROCODILIA.

Alligator mississipiensis : *Hg. crocodilinorum*, Börner, 1901, North America; Plimmer, 1912, North America.

Caiman latirostris : *Hg. caimani*, Carini, 1909, Brazil.

Caiman sclerops : *Hg.* sp., Migone, 1916, Paraguay. *Hg. brasiliensis*, Raul di Primo, 1925, Brazil.

Crocodilus cataphractus : *T.* sp., Dutton, Todd and Tobey, 1907, Belgian Congo; Plimmer, 1914, Nigeria.

(Crocodilus frontatus) = Osteolæmus tetraspis : *Hg. crocodilinorum*, Börner, 1901, North America.

Crocodilus niloticus : *T. kochi*, Laveran and Mesnil, 1912; Koch, 1906, 1909, Central Africa; Kleine, 1910, Central Africa; Kleine and Taute, 1911, Central Africa; Minchin, Gray and Tulloch, 1905, Uganda; Lloyd, Johnson, Young and Morrison, 1924, Nigeria. *Hg. pettiti*, Thiroux, 1910, 1913, Africa; Wenyon, 1909, Sudan. *Hg.* sp., Leger, M. and A., 1914, Senegal.

Crocodilus palustris : *Hg.* sp., Prowazek, 1912, Sumatra.

Crocodilus porosus : *Hg.* sp., Dobell, 1910, Ceylon.

Gavialis gangeticus : *Hg. hankini*, Simond, 1901, India.

Osteolæmus tetraspis = (Crocodilus frontatus).

AMPHIBIA (URODELA).

Batrachoseps attenuatus : *Hg. riedyi*, Eysen, 1897, California.

(Diemyctylus viridiscens) = Molge viridiscens : *T. diemyctili*, Tobey, 1906, North America; Hegner, 1920, North America.

Molge cristata : *Hg. tritonis*, Fantham, 1905, England.

Molge pyrrhogastra = (Triton pyrrhogaster).

Molge viridiscens = (Diemyctylus viridiscens).

(Triton pyrrhogaster) = Molge pyrrhogastra : *T. tritonis*, Ogawa, 1914, Japan.

AMPHIBIA (ANURA).

Bufo marinus : *Hg. cayennensis,* Leger, 1918, French Guiana. *Hg. darlingi,* Leger, 1918, French Guiana. *Hg. minima,* Leger, 1918, French Guiana. *Hg. sp.,* Darling, 1912, Panama; Plimmer, 1912, South America; Nino, 1926, Brazil.

Bufo mauritanicus : *Hg. tunisiensis,* Nicolle, 1904, Tunis.

Bufo melanostictus : *T.,* Mathis and Leger, 1911, Tonkin; Schein, quoted by Laveran and Mesnil, 1912, Annam; Donovan (first record), India. *Hg. boueti,* França, 1910, Tonkin. *Hg. tonkinensis,* Leger, 1918; Mathis and Leger, 1911, Tonkin; Prowazek, 1912, Sumatra. *Hg. bisecans,* Shortt, 1917, India. *Hg.,* Donovan (first record), and Z.S., 1925, India. *Toddia* sp., Mathis and Leger, 1912, Tonkin.

Bufo regularis : *T. mega,* Dutton and Todd, 1903, and Dutton, Todd and Tobey, 1907, Congo; Martin, Lebœuf and Roubaud, 1909, Congo; Bouet, 1909, West Africa; Macfie, 1914, Nigeria; França, 1925, Angola. *T. karyozeukton,* Dutton and Todd, 1903, and Dutton, Todd and Tobey, 1907, Congo; França, 1925, Angola. *T. rotatorium,* Balfour, 1909, Wenyon, 1909, and Stevenson, 1911, Sudan; Martin, Lebœuf and Roubaud, 1909, Congo; Bouet, 1909, West Africa; Macfie, 1914, and Lloyd, Johnson, Young and Morrison, 1924, Nigeria; Z.S., 1925, Gambia. *Hg. pestanæ,* França, 1910, Portuguese Guinea. *Hg. boueti,* França, 1910, Portuguese Guinea; França, 1925, Angola. *Hg. tunisiensis,* Macfie, 1914, Nigeria. *Hg. froilanoi,* França, 1925, Angola. *Hg.* sp. (? *Hg. boueti*), Balfour, 1909, Wenyon, 1909, and Stevenson, 1911, Sudan; Bouet, 1909, West Africa; Z.S., 1925, Gambia.

Bufo reticulatus : *T.,* Brumpt, 1906, Somaliland.

Bufo sp.: *T. mega,* Minchin, 1910, Uganda. *T.* sp., Stevenson, 1911, Sudan. *Hg. tunisiensis,* Stevenson, 1911, Sudan; Stephens, 1905, West Africa. *Hg. molœnsis,* Hoare, 1921, Uganda. *Hg.* sp., Hoare, 1921, Uganda.

Bufo vulgaris : *T.,* Grassi, 1881, 1883, Europe.

Discoglossus pictus : *T. sergenti* and *T. parroti,* Brumpt, 1923, Algiers.

Hyla arborea = (Hyla viridis) : *T. rotatorium,* Danilewsky, 1885, 1888, Europe. *T. hylæ,* França, 1908, Portugal. *T.* sp., Plimmer, 1914, Europe.

Hyla cærulea : *Hg. hylæ,* Cleland and Johnston, 1911, Australia; Cleland, 1915, Australia.

(Hyla lateristriga) = Hyla rubra : *T. borreli (T. rotatorium),* Marchoux and Salimbeni, 1907, Brazil.

Hyla lesueurii : *T. rotatorium,* Cleland and Johnston, 1911, Australia.

Hyla nasuta : *T.,* Bancroft, 1890, quoted by Johnston and Cleland, 1910, Australia.

Hyla rubra = (Hyla lateristriga).

Hyla venulosa : *T.,* Plimmer, 1912, South America.

(Hyla viridis) = Hyla arborea : *T.,* Wedl, 1850, Europe. *Cytamœba* sp., Labbé, 1894, Europe; Grassi, 1882, Italy.

Leptodactylus occellatus : *T. leptodactyli,* Carini, 1907, Brazil; Machado, 1911, Brazil; Brumpt, 1915, Brazil. *T. rotatorium,* Machado, 1911, Brazil. *Hg. leptodactyli,* Lesage, 1908, Argentine. *Hg.* sp., Carini, 1911, Brazil; Plimmer, 1917, Argentine. *Lankesterella leptodactyli,* Ducceshi, 1914, Brazil.

Leptodactylus pentadactylus : *Hg. heteronucleata,* Carini, 1909, Brazil.

Limnodynastes ornatus : *T.,* Cleland and Johnston, 1911, Australia.

Limnodynastes tasmaniensis : *T.,* Cleland and Johnston, 1911, Australia; Cleland, 1917, Australia.

Microhyla pulchra : *T.,* Mathis and Leger, 1911, Tonkin.

Pipa americana : *Hg.* sp., Leger, 1918, French Guiana.

Rana angolensis : *T.,* Laveran, 1904, South Africa. *Hg. theileri,* Laveran, 1905, Transvaal. *Hg. splendens,* Laveran, 1905, Transvaal.

Rana catesbiana : *T.,* Hegner, 1920, North America. *Hg. catesbianæ,* Stebbins, 1905, North America; Hegner, 1921, North America; Plimmer, 1915, North America. *Cytamœba bacterifera,* Hegner, 1921, North America. *Octomitus* sp., Plimmer, 1912, North America.

Rana clamata = (Rana clamitans).

(Rana clamitans) = Rana clamata : *T. rotatorium* and *T. parvum*, Kudo, 1922, North America. *T.*, Hegner, 1920, 1921, North America. *Hg. clamatœ*, Stebbins, 1905, North America; Hegner, 1921, North America; Kudo, 1922, North America. *Cytamœba bacterifera*, Hegner, 1921, North America.

Rana cyanophlyctis : *T.*, Donovan (first record), India.

Rana esculenta = (Rana ridibunda) : *T.*, Wedl, 1850, Europe; Lankester, 1882, Europe; Grassi, 1881, Europe; Danilewsky, 1885, 1888, Europe; Laveran and Mesnil, 1901, Europe; Gluge, 1842, Europe (?); Mayer, 1843, Europe (?); Gruby, 1843, Europe (?); Chaussat, 1850, Europe (?); Lieberkuhn, 1870, Europe (?); Rattig, 1875, Europe; Gaule, 1880, Europe; Sergent, Ed. and Et., 1905, Algeria; Nöller, 1912, Europe; Plimmer, 1914, Europe. *T. inopinatum*, Sergent, 1904, Algeria. *Dactylosoma splendens*, Labbé, 1894, Europe; Nöller. 1912, Europe. *Hg. splendens*, Labbé, 1894, Europe; França, 1908, Portugal, *Hg. magna*, Grassi and Feletti, 1891, Italy. *Lankesterella minima*, Nöller, 1912, Germany; Lankester, 1882, England. *Octomitus intestinalis*, Poncelle, 1919, Europe. *Cytamœba bacterifera*, Labbé, 1894, Italy.

Rana galamensis : *T.*, Dutton, Todd and Tobey, 1907, Congo. *Toddia* sp., Dutton, Todd and Tobey, 1907, Congo.

Rana guentheri : *T.*, Mathis and Leger, 1911, Tonkin. *Hg. minima*, Mathis and Leger, 1911, Tonkin. *Hg. splendens*, Mathis and Leger, 1911, Tonkin. *Hg. scheini*, Mathis and Leger, 1911, Tonkin. *Hg. theileri*, Mathis and Leger, 1911, Tonkin.

Rana hexadactyla : *T.*, Dobell, 1910, Ceylon; Patton, 1908, India; Donovan (first record), India. *Hg.* sp., Patton, 1909, India; Donovan (first record), India.

Rana limnocharis : *T.* sp., Mathis and Leger, 1911, Tonkin. *Hg. encapsulœ*, Berestneff, 1902, India. *Hg. minima (Lankesterella minima)*, Mathis and Leger. 1911, Tonkin. *Hg. splendens*, Mathis and Leger, 1911, Tonkin. *Hg. scheini*, Mathis and Leger, 1911, Tonkin. *Hg. theileri*, Mathis and Leger, 1911, Tonkin.

Rana macrodon : *T.*, Z.S., 1925, Malaya.

Rana mascariensis : *T.*, Dutton, Todd and Tobey, 1907, Congo. *Hg. neireti*, Laveran, 1906, Madagascar. *Toddia* sp., Dutton, Todd and Tobey, 1907, Congo.

Rana nutti : *T. tumida*, Awerinzew. 1918, East Africa. *Hg.*, Awerinzew, 1913, East Africa.

Rana oxyrhynchus : *T.*, Dutton, Todd and Tobey, 1907, Congo. *Toddia* sp., Dutton, Todd and Tobey, 1907, Congo.

(Rana ridibunda) = Rana esculenta : *Hg. magna*, Wülker, 1919, Macedonia.

Rana rugosa : *T.*, Koidzumi, 1911, Japan.

Rana sp. : *T. nelsprutense*, Laveran, 1904, Transvaal. *T. karyozeukton*, Martin. Lebœuf and Roubaud, 1909, Congo. *Hg. neireti*, Laveran, 1905, Madagascar. *Hg. berestneffi*, Castellani and Willey, 1905. Ceylon.

Rana temporaria : *T.*, Danilewsky, 1885 and 1888, Europe.

Rana tigrina : *T. rotatorium*, Patton, 1908, India. *T. hendersoni*, Patton, 1908, India. *Hg.* sp., Patton, 1908, India; Schein, 1910, Annam. *Hg. encapsulœ*, Berestneff, 1902, India; Patton, 1909, India. *Hg. minima (Lankesterella minima)*, Patton, 1908, India; Mathis and Leger, 1911, Tonkin. *Hg. splendens*, Mathis and Leger, 1911, Tonkin. *Hg. scheini*, Mathis and Leger, 1911, Tonkin. *Hg. theileri*, Mathis and Leger, 1911, Tonkin. *Hg. berestneffi*, Castellani and Willey, 1905, Ceylon; Patton, 1908, India. *Hg. magna*, Patton, 1908, India. *T.*, *Hg.*, and *Lankesterella minima*, Z.S., 1925, India.

Rana trinodis : *T.*, Dutton and Todd, 1903, Gambia.

Rappia marmorata : *T.*, Dutton, Todd and Tobey, 1907, Congo.

Rhacophorus leucomystax : *T.*, Mathis and Leger, 1911, Tonkin.

Xenopus lævis (clawed toad): *Herpetomonas xenopi*, Fantham, 1922, South Africa. *Hg.*, Fantham, 1919, South Africa.

PISCES.

Abramis brama (common bream): *T. abramidis*, Laveran and Mesnil, 1904, France; Keysselitz, 1906, Germany; Minchin, 1909, England. *Tp. borreli*, Keysselitz, 1906, Germany. *Tp. abramidis*, Brumpt, 1906, France; Minchin, 1909, England.

Accipenser ruthenus (sterlet): *Hg. accipenseris*, Nawrotsky, 1914, Russia.

Acerina acerina = (Acerina cernua).

(Acerina cernua) = Acerina acerina (ruff): *T. acerinæ*, Brumpt, 1906, France; Keysselitz, 1906, Germany. *Tp. borreli*, Keysselitz, 1906, Germany.

Agonus catapractus (pogge): *T. catapracti*, Henry, 1913, England. *Hg. catapracti*, Henry, 1913, England.

Anabas scandens (climbing perch): *T.*, Mathis and Leger, 1911, Tonkin.

Anguilla bengalensis = (Anguilla mauritiana).

(Anguilla mauritiana) = Anguilla bengalensis : *T. anguillicola*, Johnston and Cleland, 1910, Australia.

Anguilla reinhardtii : *T. anguillicola*, Johnston and Cleland, 1910, Australia.

Anguilla sp.: *T. granulosum*, França, 1908, Portugal. *Hg. lignieresi*, Laveran, 1906, South America. *Hg. bettencourti*, França, 1908, Portugal.

Anguilla vulgaris (eel): *T. granulosum*, Laveran and Mesnil, 1902, France; Sabrazez and Muratet, 1902, France; Manca, 1906, Sardinia; Keysselitz, 1906, Germany; França, 1907, Portugal; Minchin, 1909, England; Lebailly, 1906, France; Coles, 1914, England.

Annarrhichas lupus (catfish): *Hg. annarrhichadis*, Henry, 1912, England.

Arnoglossus grohmanni : *Globidium (Globidiellum) multifidum*, Neumann, 1909, Naples.

Arnoglossus laterna = Platophrys laterna (megrim) : *T. laternæ*, Henry, 1913, Shetland.

Auchenoglanis biscutatus : *T. simondi*, Lebœuf and Ringenbach, 1910, Congo.

Bagrus bayad (bayad): *T.*, Neave, 1906, Sudan.

Barbus barbus = (Barbus fluviatilis).

Barbus carnaticus : *T.*, Lingard, 1903, India.

(Barbus fluviatilis) = Barbus barbus (barbel): *T. barbæ*, Brumpt, 1906, France; Keysselitz, 1906, Germany. *Tp. borreli*, Keysselitz, 1906, Germany; *Tp. barbæ*, Brumpt, 1906, France.

Blennius galerita = (Blennius montagui).

Blennius gattorugine (gattorugine): *Hg. bigemina*, Henry, 1913, England.

(Blennius montagui) = Blennius galerita (Montague's blenny): *Hg. bigemina*, Laveran and Mesnil, 1901, France.

Blennius pholis (shanny): *T. delagei*, Brumpt and Lebailly, 1904, Europe. *Hg. bigemina*, Laveran and Mesnil, 1901, France; Henry, 1913, Isle of Man and England.

Blennius trigloides : *Hg. londoni*, Yakimoff, 1915, Naples.

Bothus rhombus (brill): *T. bothi*, Lebailly, 1905, France. *Hg. bothi*, Lebailly, 1905, France.

Box salpa : *T.*, Fantham, 1919, South Africa.

(Callionymus dracunculus) = Callionymus lyra : *Hg. quadrigemina*, Brumpt and Lebailly, 1904, France. *Hg. callionymi*, Brumpt and Lebailly, 1904, France.

Callionymus lyra = (Callionymus dracunculus) (dragonet): *T. callionymi*, Brumpt and Lebailly, 1904, France; Henry, 1910, England. *Hg. quadrigemina*, Henry, 1913, Isle of Man and England. *Hg. callionymi*, Henry, 1913, Isle of Man.

Cantharus blochii (sea bream): *Hg.*, Fantham, 1991, South Africa.

Cantharus emarginata (sea bream): *Hg.* (leucocytic), Fantham, 1919, South Africa.

Carassius auratus (goldfish): *T.*, Petrie, 1905, England; Thomson, 1908, England; Robertson, 1911, England; Mathis and Leger, 1911, Tonkin. *Tp. cyprini*, Robertson, 1911, England; Chalachnikov, 1888, Kharkov.

Carassius carassius =(Carassius vulgaris).

(Carassius vulgaris) =Carassius carassius (crucian carp): *T. carassii*, Mitrophanov, 1883, Europe; Keysselitz, 1906, Germany. *Tp. cyprini*, Plehn, 1903, Germany Keysselitz, 1906, Germany.

Carcharias sp.: *T. carchariasi*, Laveran, 1908, Australia.

Catastonus commersonii : *Tp. borreli*, Mavor, 1915, America.

Cestracion japonicus (Heterodontus japonicus).

Chrysichthys auratus : *T.*, Wenyon, 1909, Sudan.

Chrysophrys globiceps : *Hg.* (leucocytic), Fantham, 1919, South Africa.

Clarias angolensis : *T.*, Dutton, Todd and Tobey, 1906, Congo.

Clarias anguillaris : *T. toddi*, Bouet, 1909, Africa; Wenyon, 1909, Sudan.

Clarias gariepinus : *T.*, Fantham, 1919, South Africa.

Clarias macrocephalus : *T. clariæ*, Montel, 1905, Cochin-China; Mathis and Leger, 1911, Tonkin. *Tp. clariæ*, Mathis and Leger, 1911, Tonkin.

Clarias sp.: *T.*, Zupitza, 1909, Cameroons.

Clinus taurus : *Hg.*, Fantham, 1919, South Africa.

(Cobitis barbatula) = Nemachilus barbatula (loach): *T. barbatulæ*, Léger, L., 1904, Europe; Danilewsky, 1885, Europe; Keysselitz, 1906, Germany. *Tp. varium*, Léger, L., 1904, France; Keysselitz, 1906, Germany.

(Cobitis fossilis) =Misgurnus fossilis (giant loach): *T. cobitis*, Mitrophanov, 1883, Europe; Danilewsky, 1885, Europe.

Copidoglanis tandanus : *T. bancrofti*, Johnston and Cleland, 1910, Australia.

Cottus bubalis (father lasher): *T. cotti*, Brumpt and Lebailly, 1904, Europe. *Hg. cotti*, Brumpt and Lebailly, 1904, France; Henry, 1910, 1913, Isle of Man. *Hæmohormidium cotti*, Henry, 1910, Isle of Man.

Cottus gobio (bull-head): *T. langeroni*, Brumpt, 1906, Europe. *Tp. guernei*, Brumpt, 1905, France.

Cottus scorpius (father lasher): *Hg. cotti*, Henry, 1910, 1913, England; Bentham, 1917, England. *Hæmohormidium cotti*, Henry, 1910, Isle of Man.

Cyprinus carpio (common carp): *T. danilewskyi*, Laveran and Mesnil, 1904, Europe; Keysselitz, 1906, Germany. *Tp. borreli*, Keysselitz, 1906, Germany.

Dentex argyrozona : *T.*, Fantham, 1919, South Africa. *Hg.* (leucocytic), Fantham, 1919, South Africa. *Herpetomonas denticis*, Fantham, 1919, and Fantham and Porter, 1920, South Africa.

Diagramma mediterraneum : *Hg. dabarensis*, Leger, A. and M., 1920, Dakar.

Doras armatulus : *Hg.*, Migone, 1916, Paraguay.

Esox lucius (pike): *T. remaki*, Laveran and Mesnil, 1901, France; Danilewsky, 1885, Europe; Chalachnikov, 1888, Europe; Keysselitz, 1906, Germany; Minchin, 1909, England; Nawrotzky, 1914, Europe; Coles, 1914, England. *Tp. borreli*, Keysselitz, 1906, Germany. *Tp. gurneyorum*, Minchin, 1909, England; Nawrotzky, 1914, Russia. *Hg. esoci*, Nawrotzky, 1914, Russia.

Esox reticulatus =(Lucius reticulatus) (pickerel): *T. remaki*, Kudo, 1921, North America.

Etroplus maculatus : *T.*, Patton, 1908, India.

(Flesus vulgaris) =Pleuronectes flesus (flounder): *Hg. flesi*, Lebailly, 1904, France.

Gadus æglefinus (haddock): *T. æglefini*, Henry, 1913, Shetland. *Hg. æglefini*, Henry, 1913, Shetland. *Globidium (Globidiellum) multifidum*, Neumann, 1909, Naples; Henry, 1913, Isle of Man.

Gadus pollachius (pollack): *Hg. pollachii*, Henry, 1913, Isle of Man.

Gobio fluviatilis =Gobio gobio (gudgeon): *T. elegans*, Brumpt, 1906, France. *Tp.*, Laveran and Mesnil, 1912, France. *Hg. gobionis*, (?) Franchini, 1923, France.

Gobio gobio =Gobio fluviatilis.

(Gobius auratus) =Gobius parnelli (little and speckled goby): *Hg. hartochi*, Yakimoff, 1915, Naples.

Gobius capito (giant goby): *Hg. yakimowi-kohli*, Wladimirow, 1910 (quoted by Yakimoff, 1915), and Yakimoff, 1915, Naples.

Gobius cruentatus : *Hg. wladimirovi*, Yakimoff, 1915, Naples.

Gobius giuris : *T.*, Castellani and Willey, 1905, Ceylon; Patton, 1908, India.

(Gobius jozo) =**Gobius niger** : *Hg. marzinowskii*, Yakimoff, 1915, Naples.

Gobius minutus (spotted goby): *Hg. minuta*, Neumann, 1908 and 1909, Naples. *Globidium (Globidiellum) multiforme*, Neumann, 1909, Naples.

Gobius niger =**(Gobius jozo)** (rock goby): *T. gobii*, Brumpt and Lebailly, 1904, Europe. *Hg. blanchardi*, Brumpt and Lebailly, 1904, France. *Hg. gobii*, Brumpt and Lebailly, 1904, France.

Gobius nudiceps : *T. nudigobii, T. capigobii*, Fantham, 1919, South Africa.

Gobius paganellus (paganellus): *Hg. polypartita*, Neumann, 1908 and 1909, Naples; Henry, 1913, England.

Gobius parnelli =**(Gobius auratus).**

(Heterodontus japonicus) =**Cestracion japonicus** : *Hg. heterodonti*, Prowazek, 1910, Japan.

Hippoglossoides platessoides =**(Limanda platessoides).**

(Hypostomus auroguttatus) =**Plegostomus auroguttatus** : *T. hypostomi*, Splendore, 1910, Brazil.

Idus melanotus =**(Leuciscus idus).**

(Labeo falcifer) =**Labeo falcipinnis** : *T.*, Rodhain, 1907, Congo.

Labeo falcipinnis =**(Labeo falcifer).**

Labeo macrostoma : *T.* and *Tp.*, Rodhain, 1907, Congo.

Labrus maculatus (ballan wrasse): *Hg. labri*, Henry, 1910 and 1913, Isle of Man.

(Leuciscus cephalus) =**Squalus cephalus** : *T.* and *Tp. borreli*, Keysselitz, 1906, Germany.

(Leuciscus erythrophthalmus) =**Scardinius erythrophthalmus** : *T.* and *Tp. borreli*, Keysselitz, 1906, Germany.

(Leuciscus idus) =**Idus melanotus** : *T.* and *Tp. borreli*, Keysselitz, 1906, Germany.

(Leuciscus rutilus) =**Rutilus rutilus** (roach): *T. leucisci*, Coles, 1914, England. *T.* and *Tp. borreli*, Keysselitz, 1906, Germany.

Leuciscus sp. : *T. leucisci*, Brumpt, 1906, France.

Lichia amia (albicore): *T.*, Fantham, 1919, South Africa.

(Limanda platessoides) =**Hippoglossoides platessoides** (long rough dab): *T. limandæ*, Brumpt and Lebailly, 1904, France.

Lota lota =**(Lota vulgaris).**

(Lota vulgaris) =**Lota lota** (burbot eel-pout): *T.*, Keysselitz, 1906, Germany; Laveran and Mesnil, 1912, Europe. *Tp. borreli*, Keysselitz, 1906, Germany.

(Lucius) =**Esox.**

(Lucius reticulatus) =**Esox reticulatus** : *T. remaki*, Kudo, 1921, North America.

(Macrodon malabaricus) =**Macrodon trahira** : *T. macrodonis*, Botelho, 1907, Brazil.

Macrodon trahira =**(Macrodon malabaricus).**

(Macrodopus viridi-auratus) =**Polyacanthus opercularis** (paradise fish): *T. pelligrini*, Mathis and Leger, 1911, Tonkin.

Macrones cavasius : *T.*, Castellani and Willey, 1905, Ceylon.

Macrones seenghala : *T.*, Lingard, 1904, India.

Malapterurus electricus (electric eel): *T.*, Rodhain, 1907, Congo.

Microstomus kitt =**(Pleuronectes microcephalus).**

Misgurnus anguillicaudatus : *T.* and *Tp.*, Tanabe, 1924, Japan.

Misgurnus fossilis =**(Cobitis fossilis).**

Monochirus luteus =**(Solea lutea).**

Monopterus javanensis : *T. roulei* and *Tp.*, Mathis and Leger, 1911, Tonkin.

Mugil sp. : *T.*, Neave, 1906, Sudan.

Mugil capito (grey mullet): *Hg.*, Fantham, 1919, South Africa.

Narcacion torpedo =Torpedo ocellata.

Nemachilus barbatula =(Cobitis barbatula).

Ophiocephalus maculatus (serpent-head): *T.*, Mathis and Leger, 1911, Tonkin.

Ophiocephalus obscurus (serpent-head): *T.*, Wenyon, 1909, Sudan. *Hg. nili,* Wenyon, 1909, Sudan.

Ophiocephalus striatus (serpent-head): *T.*, Lingard, 1903, India.

Pagrus laniarius (snapper): *Hg.*, Fantham, 1919, South Africa.

Perca fluviatilis (perch): *T. percœ,* Brumpt, 1906, France; Keysselitz, 1906, Germany; Minchin, 1909, England; Coles, 1914, England. *Tp. borreli*, Keysselitz, 1906, Germany.

Periophthalmus kœlreuteri (mud-skipper): *T.*, Zupitza, 1909, Cameroons.

Phoxinus aphya =Phoxinus phoxinus =(Phoxinus lævis).

(Phoxinus lævis) =Phoxinus phoxinus =Phoxinus aphya (minnow): *T. phoxini,* Brumpt, 1906, France. *Tp. borreli*, Léger, L., 1904, France.

Phoxinus phoxinus =(Phoxinus lævis) =Phoxinus aphya.

Pimelodus mangurus =(Zungaro mangurus).

Platessa vulgaris =Pleuronectes platessa (plaice): *T. platessœ,* Lebailly, 1904, France; Robertson, 1906, England; Henry, 1910, England. *Hg. platessœ,* Lebailly, 1904, France.

Platophrys laterna =Arnoglossus laterna : *T. laternœ,* Lebailly, 1904, France. *Hg. laternœ,* Lebailly, 1904, France.

Plecostomus auroguttatus =(Hypostomus auroguttatus).

Plecostomus punctatus : *T. chagasi,* Horta, 1910, and Horta and Machado, 1911, Brazil.

Pleuronectes flesus =(Flesus vulgaris) : *T. flesi,* Lebailly, 1904, France; Robertson, 1906, England. *Hg.*, Robertson, 1906, England; Coles, 1914, England.

(Pleuronectes microcephalus) =Microstomus kitt (smear dab): *Hg. platessœ,* Henry, 1913, Scotland.

Pleuronectes platessa =(Platessa vulgaris) (plaice): *T. platessœ,* Henry, 1913, Scotland; Robertson, 1906, England. *Hg. platessœ,* Lebailly, 1904, France; Henry, 1913, Isle of Man; Robertson, 1906, England.

Polyacanthus opercularis =(Macropodus viridi-auratus).

Polypterus sp. : *T.*, Neave, 1906, Sudan.

Pseudoplatystoma coruscans : *Hg.*, Migone, 1916, Paraguay.

Raja asterias =(Raja punctata).

Raja batis (skate): *T. rajœ,* Coles, 1914, England. *Hg.*, Coles, 1914, England; Bentham, 1915, England.

Raja capensis : *T.*, Fantham, 1919, South Africa.

Raja clavata (thornback ray): *T. rajœ,* Laveran and Mesnil, 1902, Europe.

Raja macrorhynchus (flapper skate): *T. rajœ,* Laveran and Mesnil, 1902, Europe.

Raja microcellata (painted ray): *Hg. delagei,* Robertson, 1906, England.

(Raja mosaica) = Raja undulata : *T. rajœ,* Laveran and Mesnil, 1902, Europe; *Hg. delagei*, Laveran and Mesnil, 1901, France.

Raja ocellata : *T. raiœ,* Kudo, 1923, North America.

Raja oxyrhinchus (long-nosed skate): *T. giganteum,* Neumann, 1909, Europe.

(Raja punctata) = Raja asterias : *T. rajœ,* Laveran and Mesnil, 1902, Europe; *T. variabile,* Neumann, 1909, Europe; Brumpt, 1906, Europe; Robertson, 1907 and 1909, England. *Hg. delagei,* Laveran and Mesnil, 1901, France.

Raja sp. : *T. rajœ,* Henry, 1913, Shetland.

Raja undulata = (Raja mosaica).

Rhamdia queleni : *T. rhamdiœ,* Botelho, 1907, Brazil.

Rutilus rutilus = (Leuciscus rutilus).

Saccobranchus fossilis : *T. sacchobranchi, T.,* Castellani and Willey, 1905, Ceylon.

Salmo fario : *Tp. truttœ,* Brumpt, 1906, France. *Tp. valentini*, Gauthier, 1920, France.

Scardinius erythrophthalmus (rudd): *T. scardinii*, Brumpt, 1906, France. *Tp. borreli*, Laveran and Mesnil, 1901, France; Minchin, 1909, England.

Scomber scomber (mackerel): *Hæmatractidium scombri*, Henry, 1910, Isle of Man.

Scorpæna scrofa : *Hg.*, Neumann, 1908, Naples.

Scorpæna ustulata : *T. scorpænæ*, Neumann, 1909, Europe. *Hg. scorpænæ*, Neumann, 1909, Naples.

Scylliorhinus canicula = (Scyllium canicula = Scyllium catulus).

Scylliorhinus cephalus = Squalus cephalus.

Scylliorhinus stellaris = (Scyllium stellaris).

(Scyllium canicula) = Scylliorhinus canicula (rough hound): *T. scyllii*, Laveran and Mesnil, 1902, Europe; Henry, 1910, Isle of Man.

(Scyllium catulus) = Scylliorhinus canicula (rough hound): *T. scyllii*, Coles, 1914, England. *Hg.*, Coles, 1914, England.

(Scyllium stellaris) = Scylliorhinus stellaris (nurse hound): *T. scyllii*, Laveran and Mesnil, 1902, Europe.

Siluris glanis (wels): *T.*, Keysselitz, 1906, Germany.

(Solea lutea) = Monochirus luteus: *Hg. clavata*, Neumann, 1908 and 1909, Naples; Henry, 1913, England; Yakimoff, 1915, Naples.

Solea monochir : *T. dohrni*, Yakimoff, 1911, Italy.

Solea solea = (Solea vulgaris).

(Solea vulgaris) = Solea solea (common sole): *T. soleæ*, Laveran and Mesnil, 1901, Europe; Lebailly, 1906, Europe; Henry, 1910, England; Coles, 1914, England. *Hg. simondi*, Laveran and Mesnil, 1901, France; Henry, 1913, Isle of Man and England; Coles, 1914, England.

Squalus cephalus = Scylliorhinus cephalus : *T. squalii*, Brumpt, 1906, France.

Syngnathus acus (greater pipe fish): *T. yakimovi*, Wladimiroff, 1910, Europe.

Synodontis notatus : *T. synodontis*, Lebœuf and Ringenbach, 1910, Congo.

Synodontis schall : *T.*, Neave, 1906, Sudan.

Synodontis sp.: *T.*, Wenyon, 1909, Sudan.

Thalassina columna : *T.* sp., Wurtz and Thiroux, 1909, Senegal.

(Tilapia lata) = Tilapia melanopleura : *T.*, Leger, M. and A., 1914, Niger. *Hg. tilapiæ*, Leger, M. and A., 1914, Senegal.

Tilapia melanopleura = (Tilapia lata).

Tilapia zillii : *T.*, Wenyon, 1909, Sudan.

Tinca tinca = Tinca vulgaris (tench): *T. tincæ*, Laveran and Mesnil, 1904, France; Danilewsky, 1885, Europe; Doflein, 1901, Europe; Keysselitz, 1906, Germany; Minchin, 1909, England. *Tp. borreli*, Keysselitz, 1906, Germany. *Tp. keysselitzi*, Minchin, 1909, England. *Hg. laverani*, (?) Franchini, 1923, France.

Tinca vulgaris = Tinca tinca.

Torpedo marmorata : *T. torpedinis*, Sabarez and Muratet, 1908, Europe. *Hg. lo-bianci*, Yakimoff, 1915, Naples.

Torpedo ocellata = Narcacion torpedo : *Hg. torpedinis*, Neumann, 1908 and 1909, Naples.

Trachinus vipera (viper weever): *Hg.*, Coles, 1914, England.

Trichogaster fasciatus : *T.*, Lingard, 1904, India. *Hg.* sp., Plimmer, 1914, India.

(Trigla corax) = Trigla pœciloptera (tubfish): *T. triglæ*, Neumann, 1909, Europe.

Trigla gurnardus (Bloch's gurnard): *T. triglæ*, Henry, 1913, Shetland.

Trigla lineata (streaked gurnard): *T. triglæ*, Minchin and Woodcock, 1910, Italy. *Hg. rovignensis*, Minchin and Woodcock, 1910, Italy.

Trigla pœciloptera = (Trigla corax).

Zeugopterus punctatus (topknot): *T.*, Henry, 1910, Isle of Man. *Hg. zeugopteri*, Henry, 1910 and 1913, Isle of Man.

Zoarces viviparus (viviparous blenny): *Hg. bigemina*, Bentham, 1917, England.

(Zungaro mangurus) = Pimelodus mangurus : *Hg.*, Migone, 1916, Paraguay.

II. INVERTEBRATE HOSTS OF TRYPANOSOMIDÆ.

The following abbreviations are used: *C.=Crithidia ; H.=Herpetomonas; L.=Leptomonas; T.=Trypanosoma*. The list contains the hosts of Trypanosomidæ of the genera *Leptomonas, Crithidia,* and *Herpetomonas*. These names are used according to the definition of the genera given above (p. 318). The first name given to a parasite is the one the flagellate should have according to this definition. If the original description has recorded leptomonas forms, but no crithidia or trypanosome forms, the flagellate is placed in the genus *Leptomonas ;* if crithidia forms and no trypanosome forms, in the genus *Crithidia ;* and if trypanosome forms, in the genus *Herpetomonas*. The first name, therefore, gives definite information regarding the known morphology of the flagellate.

INSECTA.

ORTHOPTERA:

> **Periplaneta orientalis** : *L. periplanetæ =H. periplanetæ*, Laveran and Franchini, 1920, Italy.

NEUROPTERA:

> **Panorpa communis** : *H.* sp. =*L.* sp., Zotta, 1925, Roumania.

RHYNCHOTA:

PENTATOMIDÆ:

> **Aspongopus viduatus** : *L. aspongopi*, Woodcock, 1914.   =*H. aspongopi*, Aders, 1909, Sudan.
> **Carbula jipensis** : *L.* sp., Robertson, 1912, Uganda.
> **Cœnus delius** : *H.* sp. =*L.* sp., Glasgow, 1914, America.
> **Erthesina fullo** : *L.* sp., Villain, 1925, Honan, China.   =*C.*, Carter, 1911, India.
> **Eurydema ornatum** =(Pentatoma ornata).
> **Eurydema ornatum var. pectorale** =(Pentatoma ornata var. pectoralis).
> **Graphosoma lineatum var. italicum** : *C.* sp., Franchini, 1922, Italy.
> **Holcostethus limbolarius** =(Peribalus limbolarius).
> **Pentatoma juniperina** (salivary glands and gut): *C.* sp., Franchini, 1922, Italy.
> **(Pentatoma ornata)** =Eurydema ornatum (salivary glands and gut): *C.* sp., Franchini, 1922, Italy.
> **(Pentatoma ornata var. pectoralis)** =Eurydema ornatum var. pectorale (salivary glands and gut): *C.* sp., Franchini, 1922, Italy.
> **Pentatoma sacastia** : *C.* sp., Franchini, 1922, Italy.
> **(Peribalus limbolarius)** =Holcostethus limbolarius (salivary gland only): *L.* sp. =*H.* sp., Glasgow, 1914, America.
> **Podosius maculiventris** : *H.* sp. =*L.* sp., Glasgow, 1914, America.

COREIDÆ:

> **Genus (?)**: *L.*, (?) Sant' Anna, 1913, Principe.
> **Cletus bisbipunctatus** : *L.* sp., Rodhain, Pons, Vandenbranden and Bequaert, 1913, Belgian Congo.   =*H.* sp., Drbohlav, 1925.
> **Cletus lituripennis** : *L.* sp., Rodhain, Pons, Vandenbranden and Bequaert, 1913, Belgian Congo.

RHYNCHOTA—*Continued:*

COREIDÆ—*continued:*

Chariesterus cuspidatus : *L.*, Strong, 1924, Central America (see p. 383).

Cletus varius : *C. cleti*, Hindle and Lewis, 1912, South Africa; Woodcock, 1914.

(Leptocoris trivittatus) = Serinetha trivittata : *C. leptocoridis*, McCulloch, 1915, California.

Leptoglossus membranaceus : *C.* sp., Robertson, 1912, Uganda. = *H. leptoglossi*, Drbohlav, 1925.

Mirperus jaculus : *L. mirperi*, Rodhain, Pons, Vandenbranden and Bequaert, 1913, Belgian Congo. = *H. mirperi*, Drbohlav, 1925.

Oncopeltus cingulifer : *L.* sp., Noguchi, 1924, Honduras.

Oncopeltus famelicus : *L.* sp., Rodhain, Pons, Vandenbranden and Bequaert, 1913, Belgian Congo.

Oncopeltus fasciatus (salivary glands): *L.* = *H.*, Holmes, 1925, Maryland and New Jersey. = (?) *Phytomonas elmassiani of Asclepias syriaca* (see p. 391).

Serinetha amicta : *L.* sp., Rodhain, Pons, Vandenbranden and Bequaert, 1913, Belgian Congo. = *H.* sp., Drbohlav, 1925.

Serinetha fraterna : *L. serinethœ*, Rodhain, Pons, Vandenbranden and Bequaert, 1913, Belgian Congo. = *H. serinithœ*, Drbohlav, 1925.

Serinetha trivittata = (Leptocoris trivittatus).

LYGÆIDÆ:

Anoplocnemis sp.: *C.* sp., Shortt, 1923, India.

Diuches humilis : *L.* sp., Bouet and Roubaud, 1911, French Sudan. = Developmental forms of *Phytomonas davidi* (?).

Lygæus familiaris : *L. familiaris*, Zotta,1923, Roumania.

Lygæus hospes : *L. inhospes* = *H. inhospes*, Donovan, 1909, India.

Lygæus militaris : *L. lygœi*, Woodcock, 1914. = *H. lygœi*, Patton, 1908, India; Archibald, 1911, Sudan.

Lygæus saxatilis : *L.* sp. = *H.* sp., Franchini, 1922, Italy.

Nysius euphorbiæ : *L.* sp., Lafont, 1911, Mauritius. = Developmental forms of *Phytomonas davidi* (?).

Oxycarenus lavateræ : *C. oxycareni*, Franchini, 1922, Italy.

Pselliopus zebra : *C.* sp., Tejera, 1919, Venezuela; Gonzales Rincone, 1924, Venezuela.

PYRRHOCORIDÆ:

Dysdercus casiatus : *L.* sp. = *H.* sp., Robertson, 1912, Uganda. = *H. dyscerı* and *C. dyscerci*, Drbohlav, 1925.

Dysdercus cingulatus : *L.* sp., Donovan (first record), India.

Dysdercus superstitiosus : *L.* sp., Blacklock, 1923, Sierra Leone.

Euryophthalmus convivus : *C. euryophthalmi*, McCulloch, 1917, California.

Pyrrhocoris apterus : *L. pyrrhocoris* = *H. pyrrhocoris*, Galli-Valerio, 1915, 1920, Europe; Zotta, 1912, 1921, 1923, Roumania; Léger and Duboscq, 1910, France; Franchini, 1922, Italy.

REDUVIIDÆ:

Apiomerus elatus : *C.* sp., Gonzales Rincone, 1924, Venezuela.

Apiomerus nalipa (= lanipes ?) : *C. nalipi*, Tejera, 1919, Venezuela.

(Conorhinus rubrofasciatus) = Triatoma rubrofasciata : *C. conorhinœ*, Donovan, 1909, India.

Cosmolestes pictus : *L.* sp., Rodhain, Pons, Vandenbranden and Bequaert, 1913, Belgian Congo. = *H.* sp., Drbohlav, 1925.

Hammatocerus cinctipes : *C.* sp., Tejera, 1919, Venezuela.

(Harpactor iracundus) = Rhinocoris iracundus: *L. agilis*, Chatton, 1909, France.

RHYNCHOTA—*Continued:*

    REDUVIIDÆ—*continued:*

Hiranetis braconiformis: *L.* sp., Tejera, 1919, Venezuela. *C.* sp., Gonzales Rincone, 1924, Venezuela.

Leogorrus litura: *C. lituræ*, Tejera, 1919, Venezuela; Gonzales Rincone, 1924, Venezuela.

Notocyrtus foveatus: *L. foveati (L. striati)*, Tejera, 1919, Venezuela.

Rhinocoris albopilosus: *C. vacuolata*, Rodhain, Pons, Vandenbranden and Bequaert, 1913, Belgian Congo.

Rhinocoris iracundus =(Harpactor iracundus).

Rhodnius prolixus: *H. rangeli*, Tejera, 1920, South America.

Triatoma protracta: *H. triatomæ* = *T. triatomæ*, Kofoid and McCulloch, 1916, California.

Triatoma rubrofasciata =(Conorhinus rubrofasciatus).

Zelus janus: *L.* sp., Tejera, 1919, Venezuela. *C.* sp., Gonzales Rincone, 1924, Venezuela.

  CAPSIDÆ:

Capsid sp. (?): *L.* or *C.*, Robertson, 1912, Uganda.

  HYDROMETRIDÆ:

Gerris fossarum: *C. gerridis*, Patton, 1908, India; Dunkerly, 1913, England; Woodcock, 1914, England; Fantham, 1919, South Africa. =*H. gerridis*, Doflein, 1916.

Gerris marginatus: *C. gerridis*, Becker, 1923, North America.

Gerris najas: *C.* sp., França, 1920, Portugal.

Gerris paludum: *C. gerridis*, Porter, 1910, England.

Gerris remigis: *C. gerridis*, Becker, 1923, North America (trypanosome stages seen =(?) *H. gerridis*).

Gerris rufoscutellatus: *C. gerridis*, Becker, 1923, North America.

Gerris sp.: *C. gerridis*, Rodhain, Pons, Vandenbranden and Bequaert, 1913, Belgian Congo.

Microvelia americana: *C. gerridis*, Becker, 1923, North America.

Microvelia sp.: *C. gerridis*, Patton, 1913, India.

Peritopus sp.: *C. gerridis*, Patton, 1913, India.

Velia currens: *C. veliæ*, Dunkerly, 1913, Ireland. *L. veliæ*, Dunkerly, 1913, Ireland.

  NEPIDÆ:

Nepa cinerea: *L. jaculum*, Woodcock, 1914. =*H. jaculum*, Léger, L., 1902, France; Porter, 1909, England; Léger and Duboscq, 1910.

  NAUCORIDÆ:

Naucoridæ: *C.* sp., Robertson. 1911, England.

Naucoris maculatus: *L. naucoridis*, Poisson, 1925, France.

Naucoris viridis: *L.* sp., Poisson, 1925, France.

ANOPLURA:

Pediculus vestimenti: *L. pediculi* =*H. pediculi*, Fantham, 1912, England. *C.*, Mackie, 1907, India.

Polyplax spinulosus (Hæmatopinus spinulosus): *C. hæmatopinæ*, Patton 1908, England.

TRICHOPTERA:

Anabolia sp. (larva): *C. campanulata*, Léger, L., 1903, France; Mackinnon, 1911, Scotland. =*H. campanulata*, Doflein, 1916.

Limnophilus sp. (larva): *C. campanulata*, Léger, L., 1903, France; Mackinnon, 1911, Scotland. =*H. campanulata*, Doflein, 1916.

LEPIDOPTERA:

Agrotis pronubana (body cavity): *L. chattoni*, Paillot, 1923, Europe.
Bombyx mori : *L. bombycis* = *H. bombycis*, Levaditi, 1905, France.

DIPTERA:

TIPULIDÆ (PTYCHOPTERIDÆ):

Ptychoptera contaminata (larva): *C. campanulata*, Léger and Duboscq, 1909, France. = *H. campanulata*, Doflein, 1916.

CHIRONOMIDÆ:

Chironomus plumosus (larva): *C. campanulata*, Léger, L., 1903, France; Woodcock, 1914. = *H. campanulata*, Doflein, 1916.
Forcipomyia sp.: *L.*, (?) Townsend, 1915, Peru.
Tanypus sp. (larva): *L. gracilis* = *H. gracilis*, Léger, L., 1903, France.

SIMULIIDÆ:

Simulium columbaczense : *C. simuliœ*, Georgewitch, 1909, Serbia.
Simulium sp.: *L.* sp. = *H.* sp., Robertson, 1913, Uganda.

PSYCHODIDÆ:

Phlebotomus minutus : *Bodo phlebotomi*, Shortt, 1925. = *H. phlebotomi*, Mackie, 1914, India; Patton, 1919, 1920 (see p. 435).
Phlebotomus papatasi: *L. papatasii* (? *Leishmania tropica*) = *H. phlebotomi*, Laveran and Franchini, 1920, Italy; Patton, 1919, 1920 (see p. 435). = *H. papatasii*, Adler, 1925, Palestine; Adler and Theodor, 1925, Palestine.
Phlebotomus sp.: *L. papatasii* (? *Leishmania tropica*) = *H.* sp., Wenyon, 1912, Aleppo.

CULICIDÆ:

Aëdes triseriatus = (Culex triseriatus).
Aëdes vexans = (Culex sylvestris).
Anopheles maculipennis : *C. fasciculata*, Léger, L., 1902, France; Léger and Duboscq, 1902; Joyeux, 1918, and Wenyon, 1921, Macedonia; Laveran and Franchini, 1913, Italy. *H.* sp. = flagellates, Laveran and Franchini, 1920, Italy.
Anopheles maculipennis (larva and nymph): *L.* sp. = *H.* sp., Ed. and Et. Sergent, 1906, and Et. Sergent, 1925, Algeria.
Anopheles mauritianus = (Myzorhynchus paludis).
Anopheles nili = (Myzomyia nili) : *C.* sp. = *H.* sp., Wenyon, 1909, Sudan. = *H. myzomyiœ*, Drbohlav, 1925.
Anopheles sp. (flagellates): (?) *L.* or *C.*, Ross, 1898 and 1906, India; Christophers, 1901, West Africa; Chaterjee, 1901, India.
Anopheles stephensi = (Neocellia stephensi): *C.* or *L.*, Patton, 1912, India. = *C. neoceliœ*, Drbohlav, 1925.
Anopheles superpictus : *C. fasciculata*, Wenyon, 1921, Macedonia.
Anopheles umbrosus : *C.*, Thomson and Robertson, 1925, Federated Malay States.
Culex fatigans : *L. culicus*, Woodcock, 1914. = *H. culicis*, Patton, 1912, Madras. = *H.*, Adie, 1907, India.
Culex pipiens : *L. fasciculata*, Woodcock, 1914; Nöller, 1917, Germany; Schulz, 1924, Germany. = *C. fasciculata*, Novy, MacNeal and Torrey, 1907, North America. = *H.* sp., Patton, 1907, England. *C. culicis*, Woodcock, 1914. = *T. culicis*, Novy, MacNeal and Torrey, 1907, North America. = *T. noctuœ*, Schaudinn, 1904, Italy. = *Spirochœta ziemannii*, Schaudinn, 1904, Italy. = *H.* sp., Laveran and Franchini, 1920, Italy. *H. algeriense*, Ed. and Et. Sergent, 1906, and Et. Sergent, 1925, Algeria. = *H.*, *L.*, and *T.*, Laveran and Franchini, 1920, Italy.

DIPTERA—*Continued:*

CULICIDÆ—*continued:*

Culex pungens : *L. fasciculata*, Woodcock, 1914. = *C. fasciculata*, Novy, MacNeal and Torrey, 1907, North America. *C. culicis*, Woodcock, 1914. = *T. culicis*, Novy, MacNeal and Torrey, 1907, North America.

(Culex richiardii) = **Tæniorhynchus richiardii** : *C. culicis*, Mezincescu, 1908, Europe.

Culex sp. (flagellates): *L.* or *C.*, (?) Ross, 1898 and 1906, India; Christophers, 1901, West Africa; Patton, 1912, India.

Culex sp. (larvæ): *C.* or *L.*, Lingard and Jennings, 1906, India.

Culex sp. (salivary glands): *C.*, Mathis, 1914, Tonkin.

(Culex sylvestris) = **Aëdes vexans** : *L. fasciculata*, Woodcock, 1914. = *C. fasciculata*, Novy, MacNeal and Torrey, 1907, North America. *C. culicis*, Woodcock, 1914. = *T. culicis*, Novy, MacNeal and Torrey, 1907, North America.

(Culex triseriatus) = **Aëdes triseriatus** : *L. fasciculata*, Woodcock, 1914. = *C. fasciculata*, Novy, MacNeal and Torrey, 1907, North America. *C. culicis*, Woodcock, 1914. = *T. culicis*, Novy, MacNeal and Torrey, 1907, North America.

(Myzorhynchus paludis) = **Anopheles mauritianus (flagellates)**: Dutton, Todd and Tobey, 1907, Congo.

(Stegomyia fasciata) = **Aedes argenteus**: *H. algeriense*, Ed. and Et. Sergent, 1906, and Et. Sergent, 1925, Algeria. Flagellates, Durham, 1900, South America. *C.* sp., Noc and Stévenel, 1913, Martinique.

(Stegomyia fasciata) = **Aëdes argenteus (larva and pupa)**: *L.* sp. = *H.* sp., Wenyon, 1911, Mesopotamia

Tæniorhynchus richiardii = **(Culex richiardii)**.

Theobaldia annulata : *L. fasciculata*, Schulz, 1924, Germany.

STRATIOMYIDÆ:

Stratiomyia chameleon : *L. stratiomyiæ* = *H. stratiomyiæ*, Fantham and Porter, 1913, England.

Stratiomyia potamida : *L. stratiomyiæ* = *H. stratiomyiæ*, Fantham and Porter, 1913, England.

Stratiomyia sp. (larva): *L. larvicola*, Roubaud, 1909, Congo. = *H. larvicola*, Drbohlav, 1925.

TABANIDÆ:

Chrysops funebris : *C.* sp., Fraser and Duke, 1912, Uganda.

Hæmatopota duttoni : *C. tenuis*, Rodhain, Pons, Vandenbranden and Bequaert, 1913, Belgian Congo.

Hæmatopota italica : *C. subulata* = *H. subulata*, Léger, L., 1902, France.

Hæmatopota pluvialis : *C. hæmatopotæ*, Jegen, 1924, Switzerland. = *C.* sp., Knuth and Rauchbaar, 1910, Germany.

Hæmatopota vandenbrandeni : *C. tenuis*, Rodhain, Pons, Vandenbranden and Bequaert, 1913, Belgian Congo.

Hæmatopota sp.: *C.* sp., Donovan (first record), India.

Pangonia australis : *C.* sp., Monfallet, 1913, Chili.

Pangonia infusca : (erroneously used for *P. neavei* by Rodhain, Pons, Vandenbranden and Bequaert, 1912, and Rodhain, 1913).

Pangonia neavei : *C. pangoniæ*, Rodhain, Pons, Vandenbranden and Bequaert, 1913, and Rodhain, 1913, Belgian Congo. = *L. pangoniæ*, Rodhain, Pons, Vandenbranden and Bequaert, 1912, Belgian Congo. = *L.*, Rodhain, Bequaert, Pons and Vandenbranden, 1911, Belgian Congo.

Tabanus africanus : *C.* sp. = *H.* sp., Wenyon, 1909, Sudan.

Tabanus bovinus : *C.* sp., Nöller, 1915, Poland.

Tabanus bromius : *C.* sp., Nöller, 1916, Berlin.

DIPTERA—*Continued :*

TABANIDÆ—*continued :*

Tabanus congoiensis : *L.* sp. =*H.* sp., Bruto da Costa, 1914; Sant' Anna, 1915, Isle of Principe.

Tabanus ditæniatus : *C.* sp. =*H.* sp., Wenyon, 1909, Sudan.

Tabanus fasciatus : *C.* sp. =*H.* sp., Wenyon, 1909, Sudan.

Tabanus fuscomarginatus : *C.* sp., Bruce, Hamerton and Bateman, 1910, Uganda.

Tabanus glaucopis : *C. subulata* =*H. subulata*, Léger, L., 1904, France; Doflein, 1916. = *T. theileri*, Nöller, 1916, Germany (see p. 501).

Tabanus gracilis : *C.* sp. =*H.* sp., Wenyon, 1909, Sudan.

(Tabanus hilaris) =Tabanus tenens : *C. tabani*, Patton, 1908, India.

Tabanus par : *C.* sp. =*H.* sp., Wenyon, 1909, Sudan.

Tabanus secedens : *C.* sp., Bruce, Hamerton and Bateman, 1910, Uganda; Fraser and Duke, 1912, Uganda.

(Tabanus socius) =Tabanus tæniola : *C.* sp. = *H.* sp., Wenyon, 1909, Sudan.

Tabanus sp. : *C. tabani*, Patton, 1908, India; Mathis and Leger, 1911, Tonkin. *C.* sp., Knuth and Rauchbaar, 1910, Germany.

Tabanus striatus : *C.* sp., Patton, 1910, India.

Tabanus tæniola =(Tabanus socius =Tabanus virgatus) : *L.* sp. =*H.* sp., Bruto da Costa, 1914; Sant' Anna, 1915, Isle of Principe.

Tabanus tenens =(Tabanus hilaris).

Tabanus tergestinus : *C. minuta*, Léger, L., 1903, France. =*H. minuta*, Léger, L., 1904, France; Doflein, 1916.

Tabanus testaceomaculatus : *C.* sp., Monfallet, 1913, Chili.

Tabanus thoracinus : *C.* sp., Bruce, Hamerton and Bateman, 1910, Uganda; Fraser and Duke, 1912, Uganda.

(Tabanus virgatus) =Tabanus tæniola : *C.* sp. =*H.* sp., Wenyon, 1909, Sudan.

ASILIDÆ :

Asilus sp. : *L.* sp., Rodhain, Bequaert, Pons and Vandenbranden, 1911, Belgian Congo.

Dioctria rufipes : *H. lepto-trypanoides*, Hollande, 1922, Europe.

SYRPHIDÆ :

Eristalis tenax (larva) : *L. eristalis* =*H. eristalis*, Fantham, 1924, South Africa.

SEPSIDÆ :

Sepsis sp. : *H.* sp., Drbohlav, 1925. =*L.* sp., Roubaud, 1912, French Sudan. *H.* sp. =*C.* sp., Patton, 1910 and 1921, Madras. *L.* sp. =*H.* sp., Patton, 1910, and 1921, Madras.

CORDYLURIDÆ :

Dryomysa anilis =Neuroctina anilis.

Neuroctina anilis =Dryomysa anilis : *L.* sp. =*H. muscæ domesticæ*, Mackinnon, 1910, England.

Scatophaga hottentota : *L.* sp. =*H.* sp., Bayon, 1915, Robben Island.

Scatophaga lutaria : *L. scatophagæ* =*H. muscæ domesticæ*, Mackinnon, 1910, England.

Scatophaga sp. : *L. scatophagæ*, Dunkerly, 1913, Ireland.

Scatophaga stercoraria : *L. scatophagæ* =*H. scatophagæ*, Galli-Valerio, 1913, Europe.

BORBORIDÆ :

Borbus sp. : *L.* sp. = *H. muscæ domesticæ*, Patton, 1921, Madras.

Limosina hirtula : *L.* sp., Chatton, 1912, France.

DIPTERA—*Continued:*

BORBORIDÆ—*continued:*

Limosina sp.: *H. calliphoræ* (?), Becker, 1923, North America.
Sphærocera subsultans: *L. legerorum*, Chatton, 1912, France.   =*H. legerorum*, Drbohlav, 1925.

DROSOPHILIDÆ:

Drosophila ampelophila: *H. ampelophilæ*, Drbohlav, 1925.   =*L. ampelophilæ*, Chatton and Leger, 1911, France.
Drosophila confusa (larva, gut and Malpighian tubes): *H.* sp. (1) = *T. drosophilæ*, Chatton and Alilaire, 1908, France. = *Rhynchoidomonas drosophilæ*, Chatton, 1913. =*C. lesnei*, Alexeieff, 1912.
Drosophila confusa (larva and adult): *H. drosophilæ*, Woodcock, 1914; Doflein, 1916; Drbohlav, 1925.   =*L. drosophilæ*, Chatton and Alilaire, 1908, France.
Drosophila confusa (adult): *H.* sp. (2) =*L.* sp., Chatton and Leger, 1912, France. *H.* sp. =*L.* (*g*), Chatton and Leger, 1912, France.
Drosophila confusa (adult, Malpighian tubes): *H. roubaudi*, Drbohlav, 1925. =*L. roubaudi*, Chatton, 1912, France.
Drosophila phalerata: *H.* sp., Drbohlav, 1925.   =*L.* sp., Chatton and Leger, 1911, France.
Drosophila rubrostriata: *H. rubrostriatæ*, Doflein, 1916; Drbohlav, 1925.   =*L. rubrostriatæ*, Chatton and Leger, 1911, France.
Drosophila sp.: *H. drosophilæ* =*L. drosophilæ*, Roubaud, 1912, French Sudan. *L.* sp. =*H. muscæ domesticæ*, Patton, 1921, Madras.
Drosophila sp. adult, Malpighian tubes: *H.* sp. =*Cercoplasma drosophilæ*, Roubaud, 1912, French Sudan.

ANTHOMYIDÆ:

Anthomyia maculata: *L. anthomyiæ* =*H. anthomyiæ*, Franchini, 1922, Italy.
Fannia canicularis =(Homalomyia canicularis): *L.* sp. =*H. muscæ domesticæ*, Patton, 1921, Madras.
Fannia scalaris =(Homalomyia scalaris).
Fucellia fucorum: *L.* sp., Chatton, 1924, France.
Graphomyia maculata: *L. graphomyiæ* =*H. graphomyiæ*, Franchini, 1922, Italy.
(Homalomyia canicularis) =Fannia canicularis.
(Homalomyia scalaris) =Fannia scalaris (adult and larva): *H. homalomyiæ*, Brug, 1914, Europe. =*H. muscæ domesticæ*, Léger, 1903.
Homalomyia sp. (corvina ?): *H. homalomyiæ* =*H. muscæ domesticæ*, Mackinnon 1910, England.

OSCINIDÆ:

Siphunculina funicola: *H. siphunculinæ* =*Rhynchoidomonas siphunculinæ*, Patton, 1921, Madras. *H.* sp. =*H. siphunculinæ*, Patton, 1921, Madras.

EPHYDRIDÆ:

Notiphila (?): *L.* sp., Patton, 1912, India.

SARCOPHAGIDÆ:

Sarcophaga bullata: *H. muscarum* =*H. muscæ domesticæ*, Becker, 1923, North America.
Sarcophaga hæmorroidalis: *L. sarcophagæ* =*H. sarcophagæ*, Prowazek, 1904, Europe; Laveran and Franchini, 1920, Italy.
Sarcophaga melanura: *L. mirabilis*, Franchini, 1922, Italy.
Sarcophaga nurus: *L. sarcophagæ* =*H. muscæ domesticæ*, Roubaud, 1909, Belgian Congo.
Sarcophaga sarraceniæ: *H. lineata*, Swingle, 1911, North America.
Sarcophaga securifera: *H. muscæ domesticæ*, Becker, 1923, North America.

DIPTERA—*Continued :*

SARCOPHAGIDÆ—*continued :*

Sarcophaga sp.: *L. sarcophagœ* =*H. sarcophagœ*, Lingard and Jennings, 1906, and Patton, 1908, 1921, India. *L. sarcophagœ*, Prowazek, 1910, Japan. =*H.* sp, Prowazek, 1912, Sumatra.

ŒSTRIDÆ:

Gedœlstia paradoxa (larva): *L.* sp. =*H.* sp., Rodhain, 1915, Belgian Congo.

Kirkia minuta (larva): *L.* sp. =*H.* sp., Rodhain, 1915, Belgian Congo.

Kirkia sp. (larva): *L.* sp. =*H.* sp., Rodhain, 1915, Belgian Congo.

Œstrus aureoargentatus (larva): *L.* sp. =*H.* sp., Rodhain, 1915, Belgian Congo.

Œstrus bertrandi (larva): *L.* sp. =*H.* sp., Rodhain, 1915, Belgian Congo.

Rhinœstrus nivarleti (larva): *L. œstrorum* =*H.* sp., Rodhain, 1915, Belgian Congo. =*H. œstrorum*, Drbohlav, 1925.

MUSCIDÆ:

Auchmeromyia luteola : *H. cauleryi* =*Cercoplasma cauleryi*, Roubaud, 1911, French Sudan.

Calliphora coloradensis : *H. calliphorœ*, Swingle, 1911, North America.

Calliphora erythrocephala : *H. muscarum* =*H. muscœ domesticœ*, Alexeieff, 1911; Becker, 1923, North America. =*Rhynchoidomonas luciliœ*, Alexeieff, 1911. =*H. gracilis*, Alexeieff, 1911. =*C. calliphorœ*, Swellengrebel, 1911, Europe. =*L. calliphorœ*, Swellengrebel, 1911, Europe. =*H. calliphorœ*, Galli-Valerio, 1914, Europe. =*H.* sp., Laveran and Franchini, 1920, Europe.

Calliphora vomitoria : *H. calliphorœ*, Galli-Valerio, 1916, Europe.

Ceroxys crassipennis : *L.* sp. =*H.* sp., Laveran and Franchini, 1920, France.

Chrysomyia albiceps =(Pycnosoma putorium) : *H. mirabilis*, Patton, 1921, Madras.

Chrysomyia macellaria : *H. muscarum* =*H. muscœ domesticœ*, Becker, 1923, North America.

(?) Chrysomyia marginale =(Pycnosoma marginale).

Chrysomyia megalocephala : *H. mirabilis*, Patton, 1921, Madras.

Cordylobia rodhaini =Stasisia rodhaini.

Dasyplura prætorum : *L. lesnei* =*H. lesnei*, Léger, 1903, Europe; Drbohlav, 1925. =*C. lesnei*, Alexeieff, 1912.

Glossina brevipalpis : *H. grayi* (probably crocodile trypanosome) =*T. grayi*, Bruce and collaborators, 1915, Africa.

Glossina morsitans : *L.* sp., Lloyd, 1924, Nigeria.

Glossina palpalis : *H. grayi* (probably crocodile trypanosome) =*T. grayi*, Novy, 1906, Africa; Minchin, 1907; Roubaud, 1909; Kinghorn and Montgomery, 1909; Kleine, 1910; Sant' Anna, 1913; Bruce and collaborators, 1915; Macfie, 1916. =*Cystotrypanosoma grayi*, Roubaud, 1912. =*C. grayi*, Drbohlav, 1925. =*T. kochi* of crocodile, Koch, 1909; Kleine, 1910; Kleine and Taute, 1911. =*T.* of bird, Minchin, 1910 (see pp. 373, 582).

Glossina tachinoides : *H. grayi* (possibly monitor trypanosome, *T. varani*), Lloyd and Johnson, 1924, Nigeria.

(Lucilia argyrocephala) =Lucilia serenissima : *L.* sp. =*H. muscœ domesticœ*, Patton, 1921, Madras. *H. mirabilis*, Patton, 1921, Madras.

Lucilia cæsar : *H. luciliœ*, Galli-Valerio, 1923, Europe. =*H.* sp., Strickland and Roy, 1925, India.

Lucilia craggi : *L.* sp. =*H. muscœ domesticœ*, Patton, 1921, Madras. *H. mirabilis*, Patton, 1921, Madras.

Lucilia latifrons : *H. mesnili*, Doflein, 1916. =*L. mesnili*, Roubaud, 1908, Belgian Congo. =*Cercoplasma mesnili*, Roubaud, 1911.

Lucilia pilatea : *H. mesnili*, Doflein, 1916. =*L. mesnili*, Roubaud, 1908, Belgian Congo. =*Cercoplasma mesnili*, Roubaud, 1911.

DIPTERA—*Continued :*

MUSCIDÆ—*continued :*

Lucilia serenissima =(Lucilia argyrocephala) (Malpighian tubes and gut): *H. luciliæ* = *Rhynchomonas luciliæ*, Patton, 1910, India. = *Rhynchoidomonas luciliæ*, Patton, 1910. = *H. muscæ domesticæ*, Patton, 1910. = *C. lesnei*, Alexeieff, 1912.

Lucilia sericata : *H. muscarum* = *H. muscæ domesticæ*, Becker, 1923, North America.

Lucilia sp.: *L. luciliæ* = *H. luciliæ*, Strickland, 1911, England.

Lucilia sp. (sericata ?): *H. intestinalis* = *Cystotrypanosoma intestinalis*, Roubaud, 1911, French Sudan. = *C. lesnei*, Alexeieff, 1913. = *Rhynchoidomonas intestinalis*, Kudicke, 1923.

Lyperosia minuta : *C.* sp., Leger, 1922, West Africa.

Lyperosia thirouxi : *C.* sp., Leger, 1922, West Africa.

Musca bezzii : *L. craggi* = *H. craggi*, Patton, 1921, Madras.

Musca corvina : *L.* sp. = *H. muscæ domesticæ*, Roubaud, 1909, Belgian Congo.

Musca domestica : *H. muscarum* = *Bodo*, Burnett, 1851 and 1852. = *Bodo muscarum*, Leidy, 1856. = *Cercomonas muscæ domesticæ*, Stein, 1878. = *Monomita muscarum*, Grassi, 1881. = *H. muscæ domesticæ*, Kent, 1881; Prowazek, 1904; Léger, 1903; Lingard and Jennings, 1906, India; Wenyon, 1911; Alexeieff, 1911; Dunkerly, 1911; Cardamatis, 1912; Woodcock, 1914; Glaser, 1922, America; Becker, 1923, America. = *C. muscæ domesticæ*, Rosenbusch, 1910; Werner, 1908. = *L. muscæ domesticæ*, Flu, 1911; Dunkerly, 1911. = *C. lesnei*, Alexeieff, 1912.

Musca humilis : *L.* sp. = *H. muscæ domesticæ*, Patton, 1921, Madras.

Musca nebulo : *L.* sp. = *H. muscæ domesticæ*, Patton, 1910, India. = *Rhynchomonas luciliæ*, Patton, 1910. = *Rhynchoidomonas luciliæ*, Patton, 1910.

Muscina stabulans ("trypanosomes"): Franchini, 1922, Italy. *H. calliphoræ* (?), Becker, 1923, North America.

Phormia regina : *H. muscarum* = *H. muscæ domesticæ*, Becker, 1923, North America.

Pollenia rudis : *L. polleniæ* = *H. polleniæ*, Galli-Valerio, 1915, France. = *H. muscæ domesticæ*, Léger, 1903.

(Pycnosoma marginale) =Chrysomyia marginale (?): *C.* sp., Fantham, 1919, South Africa.

(Pycnosoma putorium) =Chrysomyia albiceps : *L. pycnosomæ*, Roubaud, 1909, French Congo. = *H. muscæ domesticæ*, Alexeieff, 1911. = *H. pycnosomæ*, Drbohlav, 1925. *H. mirabilis* = *L. mirabilis*, Roubaud, 1908, French Congo. = *Cercoplasma mirabilis*, Roubaud, 1911. *H. sudanensis* = *L. sudanensis*, Roubaud, 1911, French Congo. *L.* sp. = *H. muscæ domesticæ*, Roubaud, 1909, French Congo. *L.* sp., Roubaud, 1909, French Congo.

Stasisia rodhaini = (?)Cordylobia rodhaini : *L.* sp., Rodhain and Bequaert, 1916, Congo.

Stomoxys calcitrans : *L. stomoxyæ*, Jegen, 1924, Switzerland. = *H.* sp., Gray, 1906, Uganda. *C. hæmatopotæ*, Jegen, 1924, Switzerland.

Stomoxys nigra : *L.* sp. = *H.* sp., Macfie, 1913, West Africa; Sant' Anna, 1915, Principe.

Stomoxys sp.: *L.* sp. = *H.* sp., Patton, 1908, India; Wenyon, 1911, Bagdad.

Theicomyza fusca : *L.* sp. = *H. muscæ domesticæ*, Léger, 1903, France.

HIPPOBOSCIDÆ:

Melophagus ovinus : *T. melophagium* (sheep trypanosome) = *C. melophagia*, Flu, 1908, and others (see p. 502).

NYCTERIBIIDÆ:

Cyclopodia sykesi : *C. nycteribiæ*, Chatton, 1909, India.

SIPHONAPTERA:

Ceratophyllus alladinis (larva and adult): *L.* sp., Patton, 1912, India. =*H. alladinis*, Drbohlav, 1925.

Ceratophyllus columbæ: *L.* sp., Nöller, 1913, 1914, Germany.

Ceratophyllus fasciatus (larva and adult): *L. pattoni*, Chatton and Delanoë, 1912, France; Laveran and Franchini, 1914, France.

Ceratophyllus gallinæ (larva and adult): *L.* sp., Nöller, 1913, 1914, Germany. =*H. gallinæ*, Drbohlav, 1925.

Ceratophyllus sciurorum: *L. debreuili* =*H. debreuili*, Brumpt, 1913, France.

Ceratophyllus sp. (lucifer ?): *L. pattoni* =*H. pattoni*, Swingle, 1911, North America.

Ctenocephalus canis: *L. ctenocephali* =*H. ctenocephali*, Fantham, 1912, England; Shortt, 1923, India. =*H. pseudoleishmania*, Brumpt, 1913. =*L.* sp., Nöller, 1912, Germany. =*Leishmania donovani*, Basili, 1910; Alvarez and da Silva, 1911; Sangiorgi, 1910. =Flagellates, Swellengrebel and Strickland, 1910; da Silva, 1913; Marzocchi, 1911; Wenyon, 1913; Shortt, 1923, India. *C. ctenocephali*, Patton and Rao, 1921, Madras (see p. 348).

Ctenocephalus felis (adult and larva, gut and Malpighian tubes): *L.* sp. =*H.* sp., Patton, 1908, 1912, India. =*H. pulicis*, Stephens and Christophers, 1908.

Ctenophthalmus agyrtes: *L. ctenophthalmi*, Mackinnon, 1909, England. =*H. ctenophthalmi*, Drbohlav, 1925.

Ctenophthalmus agyrtes (larva and adult): *C. ctenophthalmi*, Patton and Strickland, 1908, England.

(Ctenopsylla musculi) =**Leptopsylla musculi**: *L. ctenopsyllæ* =*H. ctenopsyllæ*, Laveran and Franchini, 1915, France.

Hystrichopsylla talpæ: *C. hystrichopsyllæ*, Mackinnon, 1909, England.

Leptopsylla musculi =(Ctenopsylla musculi).

Pulex irritans: *C. porteræ*, Lavier, 1921. =*C. pulicis*, Porter, 1911, England. *L. pulicis* =*H. pulicis*, Patton and Rao, 1921, Madras. =*H.* sp., Wenyon, 1912, England. =*Leishmania donovani*, Basili, 1911, Italy. =Flagellates, da Silva, 1913 (see p. 349).

Pulex sp. (braziliense ?): *L. pattoni* =*H. pattoni*, Swingle, 1911, North America.

Xenopsylla cleopatræ: *C. pulicis* (Stephens and Christophers, 1908), Balfour, 1909, Sudan. =*H.* sp., Balfour, 1906. =*H. pulicis*, Stephens and Christophers, 1908. =*C. cleopatræ*, Patton and Rao, 1921. =*C. xenopsyllæ*, Drbohlav, 1925.

HYMENOPTERA:

APIDÆ:

Apis mellifera: *C.* sp., Fantham and Porter, 1911, England.

VESPIDÆ:

Emphytus cinctus (larva): *L. emphyti* =*H. emphyti*, Hollande, 1912, Europe.

Vespa crabo: *L. vespæ*, Porter, 1909, England.

ARACHNIDA.

IXODIDÆ:

Amblyomma sp.: *L.* sp., Tejera, 1919, Venezuela.

Hæmaphysalis flava: *C. hæmaphysalidis*, Patton, 1908, India.

Hyalomma ægyptium: *C. hyalommæ*, O'Farrel, 1913, Sudan. =*H. hyalommæ*, Doflein, 1916. =Developmental form of trypanosome (?).

Rhipicephalus sanguineus: *C. christophersi*, Patton and Strickland, 1908. =*T. christophersi*, Novy, MacNeal and Torrey, 1907, India.

Ixodes ricinus: *C.*, Bishop, 1911, England. =*T. melophagium* (?).

NEMATODA.

Nematode (marine) : Chatton, 1924, France.
Trilobus gracilis : *L. bütschlii,* Kent, 1881, Europe.

MOLLUSCA.

Burnupia capensis: *L.* sp.=*H.* sp., Fantham, 1925, South Africa.
Pachylabra mœsta : *H. pachylabræ,* Mello, 1921, India.   (?) *L.*
Patella vulgata : *L. patellæ* = *H. patellæ,* Porter, 1914, England.
Unio caffer: *L.* sp.=*H.* sp., Fantham, 1925, South Africa.

PART VI

REFERENCES TO LITERATURE—PROTOZOA AND SPIROCHÆTES

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REFERENCES TO LITERATURE

PROTOZOA.

ACTON, H. W. (1918). The significance of Charcot-Leyden crystals in the fæces as an indication of amœbic colitis. *Indian Journ. Med. Res.*, VI, 157.

ACTON, H. W., & KNOWLES, R. (1914). Studies on the Halteridium parasite of the pigeon, *Hæmoproteus columbæ*, Celli and San Felice. *Ibid.*, I, 663.

ADELHEIM, R. (1924). Über Leishmaniosis infantum et canina in Riga. *Arch. Schiffs.-u. Trop.-Hyg.*, XXVIII, 367.

ADERS, W. M. (1923). Trypanosomiasis of stock in Zanzibar. *Trans. Roy. Soc. Trop. Med. & Hyg.*, XVII, 192.

ADIE, H. (1915). The sporogony of *Hæmoproteus columbæ*. *Indian Journ. Med. Res.*, II, 671.

ADIE, H. A. (1921). Preliminary note on the development of the Leishman-Donovan parasite in spleen juice and in the alimentary tract of *Cimex lectularius* (Lin.). *Indian Journ. Med. Res.*, IX, 255.

—— (1922). [Telegram announcing finding Leishman-Donovan bodies in salivary glands and ducts of *Cimex lectularius.*] *Ibid.*, IX, v.

—— (1922a). Notes on bodies observed in *Cimex rotundatus* Linne collected in a kala-azar infected area in Assam. *Ibid.*, X, 236.

—— (1924). The sporogony of *Hæmoproteus columbæ*. *Bull. Soc. Path. Exot.*, XVII, 605.

ADIE, J. R. (1906). Note on a Leucocytozoon found in *Mus rattus* in the Punjab. *Journ. Trop. Med. & Hyg.*, IX, 325.

—— (1908). Note on a parasite in the sparrow. *Indian Med. Gaz.*, XLIII, 176.

ADLER, S. (1923). Malaria in chimpanzees in Sierra Leone. *Ann. Trop. Med. & Parasit.*, XVII, 13.

—— (1924). An *Isospora* of civet cats. *Ibid.*, XVIII, 87.

—— (1924a). A note on *Plasmodium agamæ* (Wenyon, 1908). *Ibid.*, XVIII, 131.

ADLER, S., & THEODOR, O. (1925). A sporozoon of *Phlebotomus papatasii*. *Ibid.*, XIX, 309.

———— (1925a). The experimental transmission of cutaneous leishmaniasis to man from *Phlebotomus papatasii*. *Ibid.*, XIX, 365.

ALDIGÉ, E. (1920). Trypanosomiase du porc en Guinée Française. *Bull. Soc. Path. Exot.*, XIII, 792.

ALEXEIEFF, A. (1908). Sur la division de *Hexamitus intestinalis* Dujardin. *C. R. Soc. Biol.*, LXV, 402.

—— (1909). Les flagellés parasites de l'intestin des batraciens indigènes. *Ibid.*, LXVII, 199.

—— (1909a). Un nouveau Trichomonas à quatre flagelles antérieurs. *Ibid.*, LXVII, 712.

—— (1910). Sur les flagellés intestinaux des poissons marins (note préliminaire). *Arch. Zool. Exp.*, VI, N. & R., i.

—— (1910a). Sur quelques points de la structure des " Binucléates " de Hartmann. *C. R. Soc. Biol.*, LXIX, 532.

—— (1911). Notes sur les flagellés. i. Quelques flagellés intestinaux nouveaux ou peu connus. ii. Quelques flagellés communs dans les infusions. *Arch. Zool. Exp.*, VI, 491.

———— (1911a). Sur la morphologie et la division de *Bodo caudatus* (Duj.) Stein. *C. R. Soc. Biol.*, LXX, 130.

—— (1911b). Sur la division nucléaire et l'enkystement chez quelques Amibes du groupe *limax*. *Ibid.*, LXX, 455, 534, 588.

—— (1911c). Sur les " kystes de *Trichomonas intestinalis* " dans l'intestin des Batraciens. *Bull. Sci. France & Belgique.*, XLIV, 333.

—— (1911d). Sur la nature des formations dites " kystes de *Trichomonas intestinalis.*" *C. R. Soc. Biol.*, LXXI, 296.

—— (1911e). Sur les Cercomonadines intestinales de *Calliphora erythrocephala* Mg. et de *Lucilia* sp. *Ibid.*, LXXI, 379.

—— (1911f). Sur le genre *Herpetomonas* Kent. *Ibid.*, LXXI, 455.

—— (1911g). Sur la famille Cercomonadina Bütschli emend. (non Cercomonadidæ Kent). *Ibid.*, LXXI, 506.

—— (1911h). Sur la spécification dans le genre *Trichomonas* Donné. *Ibid.*, LXXI, 539.

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1418 REFERENCES TO LITERATURE

ALEXEIEFF, A. (1912). Sur les charactères cytologiques et la systématique des amibes du groupe *limax* (*Nægleria* nov. gen. et *Hartmannia* nov. gen.) et des amibes parasites des Vertébrés (*Proctamœba* nov. gen.). *Bull. Soc. Zool.* Paris, XXXVII, 55.
—— (1912*a*). Quelques remarques complémentaires sur la systématique des amibes du groupe *limax*. Sur le genre *Sappinia* Dangeard. *Ibid.*, XXXVII, 149, 157.
—— (1912*b*). Sur quelques noms de genres des flagellés qui doivent disparaître de la nomenclature pour cause de synonymie ou pour toute autre raison. Diagnoses de quelques genres récemment étudiés. *Zool. Anz.*, XXXIX, 674.
—— (1912*c*). Sur quelques protistes parasites intestinaux d'une tortue de Ceylan (*Nicoria trijuga*). *Ibid.*, XL, 97.
—— (1912*d*). Quelques remarques à propos de la spécificité parasitaire. Sur le véritable nom de *Cryptobia* (=*Trypanoplasma*) *intestinalis* et sur celui du Trypanosome pathogène des Mammifères; quelques autres questions de synonymie chez les Protozoaires. *Ibid.*, XLI, 17.
—— (1912*e*). Notes sur les Herpetomonadidæ (=Trypanosomidæ Doflein, 1911). *Arch. Zool. Exp.*, IX, N. &. R., xxix.
—— (1912*f*). Le parasitisme des Eugléniens et la phylogénie des Sporozoaires *sensu stricto*. *Ibid.*, X, N. &. R., lxxiii.
—— (1912*g*). Sur le stade flagellé dans l'évolution des Amibes *limax*. 1. Stade flagellé chez *Amœba punctata* Dangeard. *C. R. Soc. Biol.*, LXXII, 126.
—— (1913). Systématisation de la mitose dite "primitive." Sur la question du centriole (à propos de la division nucléaire chez *Malpighiella* sp.). *Arch. Protist.*, XXIX, 344.
—— (1913*a*). Recherches sur les Sarcosporidies. 1. Étude morphologique. *Arch. Zool. Exp.*, LI, 521.
—— (1914). Notes protistologiques (I-III). *Zool. Anz.*, XLIV, 193.
—— (1914*a*). Notes protistologiques (IV-VIII). *Ibid.*, XLIII, 515.
—— (1917). Mitochondries et corps parabasal chez les flagellés. *C. R. Soc. Biol.*, LXXX, 358.
—— (1917*a*). Nature mitochondriale du corps parabasal des flagellés. *Ibid.*, LXXX, 499.
—— (1917*b*). Sur la fonction glycoplastique du kinétoplaste (=kinetonucleus) chez les flagellés. *Ibid.*, LXXX, 512.
—— (1918). Sur un flagellé coprozoite, *Alphamonas coprocola* n. g., n. sp. *Arch. Zool. Exp.*, LVII, N. & R., i.
—— (1924). Sur le corps parabasal, l'axostyle et les mitochondries chez les flagellés. *Arch. Russ. Protist.*, Moscow, III, 149.
—— (1924*a*). Sur la question du noyau chez les bactéries. (Contribution à l'étude des mitochondries et des grains métachromatiques.) *Arch. Protist.*, XLIX, 396.
ALMY (1901). Nouveau cas de piroplasmose canine. *Bull. Soc. Centr. Méd. Vét.*, VIII, 375.
ALVARES, D., & PEREIRA DA SILVA, E (1910). Sobre a existencia do Kala-Azar espontaneo no câo em Lisboa. *Med. Contemp.*, Lisbon, XXVIII, 162.
—— (1911). Sobre a frequencia do Kala-Azar nos câes em Lisboa. *Ibid.*, XXIX, 197.
ANSCHÜTZ, G. (1909). Ueber den Entwicklungsgang des "*Hæmoproteus orizivoræ*" nov. spec. *Centralbl. Bakt.*, I. Abt., LI, 654.
—— (1910). Ueber Uebertragungsversuche von *Hæmoproteus orizivoræ* und *Trypanosoma paddæ*, nebst Bemerkungen über den Entwickelungsgang des ersteren. *Ibid.*, I. Abt., LIV, 328.
ANTOLISEI, E., & ANGELINI, A. (1889). Due altri casi di febbre malarica sperimentale. *Riforma Med.*. Naples, V, 1352 & 1358.
—— (1890). Nota sul ciclo biologico dell' ematozoo falciforme. *Ibid.*, VI, 320, 326 & 332.
APSTEIN (1910). *Cyclopterus lumpus*, der Seehase. Seine Fischerei und sein Mageninhalt. *Mitt. Deutsch. Seefisch.-Vereins*, No. 10.
ARAGÃO, H. DE B. (1907). Sobre o cyclo evolutivo do halteridio do pombo. (1 e 2 notas.) *Brazil-Medico*, April 15.
—— (1908). Über den Entwicklungsgang und die Übertragung von *Hæmoproteus columbæ*. *Arch. Protist.*, XII, 154.
—— (1909). Über eine neue Amöbenart *Amœba diplomitotica*. Beitrag zum Studium der Kernteilung bei den Amöben. *Mem. Inst. O. Cruz*, I, 33.
—— (1911). Beobachtungen über Hämogregarinen von Vögeln. *Ibid.*, III, 54.
—— (1913). Nota sobre as schizogonias e gametogonias dos trypanosomos. *Brazil-Medico*, XXVII, 271.
—— (1916). *Copromastix prowazeki*, n. g., n. sp. (Nota prévia). *Ibid.*, XXX, 49.
—— (1916*a*). Pesquizas sobre o "*Hæmoproteus columbæ*." *Ibid.*, XXX, 353, 361.
—— (1922). Transmissão da Leishmaniose no Brazil pelo *Phlebotomus intermedius*. *Ibid.*, I, 129.
ARAGÃO, H. DE B., & NEIVA, A. (1909). A contribution to the study of the intraglobular parasites of the lizards. Two new species of Plasmodium, *Pl. diploglossi* n. sp. and *Pl. tropiduri* n. sp. *Mem. Inst. O. Cruz*, I, 44.
ARAI, K. (1925). Beitrag zur Infektion der Maus mit *Sarcocystis tenella*. *Arch. Protist.*, L, 213.

ARCHIBALD, R. G. (1913). An interesting case of kala-azar. *Journ. R. Army. Med. Corps*, XX, 512.

——— (1914). A preliminary report on some further investigations on kala-azar in the Sudan. *Ibid.*, XXIII, 479.

——— (1922). *Trypanosoma rhodesiense* in a case of sleeping sickness from the Sudan. *Ann. Trop. Med. & Parasit.*, XVI, 339.

ARCHIBALD, R. G., & SUSU, B. (1924). A sporozoon from the spleen of a case of splenomegaly in the Sudan. *Trans. Roy. Soc. Trop. Med. & Hyg.*, XVII, 482.

ARKWRIGHT, J. A., ATKIN, E. E., & BACOT, A. (1921). An hereditary *Rickettsia*-like parasite of the bed bug (*Cimex lectularius*). *Parasitology*, XIII, 27.

ARNDT, A. (1924). Rhizopodenstudien, I. *Arch. Protist.*, XLIX, 1.

ARTAULT, S. (1898). Flore et faune des cavernes pulmonaires. *Arch. Parasit.*, I, 217.

ASHWORTH, J. H. (1923). On *Rhinosporidium seeberi* (Wernicke, 1903), with special reference to its sporulation and affinities. *Trans. Roy. Soc. Edin.*, LIII, 301.

ASHWORTH, J. H., & RETTIE, T. (1912). On a Gregarine—*Steinina rotundata*, nov. sp.—present in the mid-gut of bird fleas of the genus *Ceratophyllus*. *Proc. Roy. Soc.*, B, LXXXVI, 31.

AUERBACH, M. (1910). *Die Cnidosporidien (Myxosporidien, Actinomyxidien, Microsporidien)*. Eine monographische Studie. Leipzig.

AUSTEN, E. E. (1903). *A monograph of the tsetse flies (genus Glossina Westwood) based on the collection in the British Museum*. London.

AVARI, K. B. C. R., & MACKIE, F. P. (1924). Canine leishmaniasis in Bombay. *Indian Med. Gaz.*, LIX, 604.

AWERINZEW, S. (1909). Studien über parasitische Protozoen. I. Die Sporenbildung bei *Ceratomyxa drepanopsettæ mihi*. *Arch. Protist.*, XIV, 74.

——— (1909a). Studien über parasitische Protozoen. IV. Beobachtungen über die Entwicklungsgeschichte von Coccidien an dem Darme von *Cerebratulus* sp. *Ibid.*, XVIII, 11.

——— (1914). Beiträge zur Morphologie und Entwicklungsgeschichte der Protozoen von Deutsch-Ost-Africa. *Journ. Microbiol.*, Petrograd, I, Nos. 1-2.

AWERINZEW, S., & MUTAFOWA (1914). Material zur Kenntnis der Infusorien aus dem Magen der Wiederkäuer. *Arch. Protist.*, XXXIII, 109.

BABES, V. (1888). Sur l'hémoglobinurie bactérienne du bœuf. *C. R. Acad. Sci.*, CVII, 692.

——— (1889). Die Aetiologie der seuchenhaften Hämoglobinurie des Rindes. *Arch. Path. Anat.*, CXV, 81.

——— (1890). Sur les microbes de l'hémoglobinurie du bœuf. *C. R. Acad. Sci.*, CX, 800.

——— (1890a). Expériences relatives à la transmissibilité de l'hémoglobinurie aux animaux. *Ibid.*, CX, 975.

——— (1892). L'étiologie d'une enzootie des moutons dénommée " Carceag " en Roumanie. *Ibid.*, CXV, 359.

BACH, F. W. (1923). Zur Kenntnis der bei Affen vorkommenden Entamöben. *Arch. Schiffs.-u. Trop.-Hyg.*, XXVII, 31.

BACOT, A. W. (1916). *Report of the entomological investigation undertaken for the* [Yellow Fever (West Africa)] *Commission for the year August*, 1914, *to July*, 1915. IV.—Parasites. III, 127.

——— (1921). Rickettsia in the bed bug: Demonstration. *Trans. Roy. Soc. Trop. Med. & Hyg.*, XIV, 92.

BAELZ, E. (1883). Ueber einige neue Parasiten des Menschen. *Berlin. Klin. Woch.*, XX, 234.

BAETJER, W. A., & SELLARDS, A. W. (1914). The behaviour of amœbic dysentery in lower animals and its bearing upon the interpretation of the clinical symptoms of the disease in man. *Bull. Johns Hopkins Hosp.*, XXV, 237.

BALBIANI, E. G. (1884). *Leçons sur les Sporozoaires*. Paris.

——— (1885). Sur un infusoire cilié parasite du sang de l'aselle aquatique (*Anoplophrya circulans*). *Recueil Zool. Suisse*, II, 277.

——— (1889). Sur trois entophytes nouveaux du tube digestif des Myriapodes. *Journ. Anat. & Physiol.*, Paris, XXV, 5.

BALDREY, F. S. H. (1906). Some problems in sheep diseases. *Journ. Trop. Vet. Sci.*, I, 387.

——— (1909). Versuche und Beobachtungen über die Entwicklung von *Trypanosoma lewisi* in der Rattenlaus *Hæmatopinus spinulosus*. *Arch. Protist.*, XV, 326.

——— (1910). Transmission of surra. *Journ. Trop. Vet. Sci.*, V, 595.

——— (1911). The evolution of *Trypanosoma evansi* through the fly: *Tabanus* and *Stomoxys*. *Ibid.*, VI, 271.

BALFOUR, A. (1905). A hæmogregarina of mammals, *H. jaculi* (*H. balfouri* Laveran). *Journ. Trop. Med. & Hyg.*, VIII, 240.

——— (1905a). A third stage in the sexual cycle of the hæmogregarine of jerboas. *Brit. Med. Journ.*, I, 1330.

BALFOUR, A. (1906). A hæmogregarine of mammals and some notes on trypanosomiasis in the Anglo-Egyptian Sudan. *Journ. Trop. Med. & Hyg.*, IX, 81.

—— (1906a). Herpetomonas parasites in fleas. *Journ. Hyg.*, VI, 652.

—— (1906b). A leucocytozoon of mammals. *2nd Rept., Wellcome Trop. Res. Labs., Khartoum*, 110.

—— (1909). Trypanosomiasis in the Anglo-Egyptian Sudan. *3rd Rept., Wellcome Trop. Res. Labs., Khartoum*, 27.

—— (1909a). Hæmogregarine of the jerboa. *Ibid.*, 35.

—— (1911). Fallacies and puzzles in blood examination. *4th Rept., Wellcome Trop. Res. Labs., Khartoum*, A, 109.

—— (1913). A sarcocyst of a gazelle (*G. rufifrons*), showing differentiation of spores by vital staining. *Parasitology*, VI, 52.

—— (1916). On the occurrence of Herpetomonads (?) in gerbils. *Ibid.*, VIII, 260.

BALFOUR, A., & WENYON, C. M. (1914). The so-called *Plasmodium tenue* (Stephens). *Journ. Trop. Med. & Hyg.*, XVII, 353.

BARABAN, L., & St. REMY, G. (1894). Sur un cas de tubes psorospermiques observés chez l'homme. *C. R. Soc. Biol.*, X, 201.

BARLOW, N. (1915). Craigiasis. *Amer. Journ. Trop. Dis. & Prev. Med.*, II, 680.

BARRET, H. P., & SMITH, N. M. (1923). The cultivation of Endamœba from the intestine of a turtle. *Amer. Journ. Hyg.*, III, 205.

—— —— (1924). The cultivation of an Endamœba from the turtle, *Chelydra serpentina. Ibid.*, IV, 155.

BARRET, H. P., & YARBROUGH, N. (1921). A method for the cultivation of *Balantidium coli. Amer. Journ. Trop. Med.*, I, 161.

BARZILAI-VIVALDI, G., & KAUDERS, D. (1924). Unübertragbarkeit alter Impfmalariastämme durch Anophelen. *Zeitschr. Hyg. u. Infekt.*, CIII, 744.

BASILE, C. (1910). Alcune osservazioni sulla presenza di Leishmanie nei cani. *Atti R. Accad. Lincei, Rendic.*, XIX, i, 158.

—— (1910a). Sulla Leishmaniosi del cane e sull' ospite intermedio del Kala-Azar infantile. *Ibid.*, XIX, ii. 523.

—— (1911). Sulla trasmissione della Leishmaniosi. *Ibid.*, XX, i, 50.

—— (1911a). Sulla Leishmaniose e sul suo modo di trasmissione. *Ibid.*, XX, i, 278, 479, 955; ii, 72.

—— (1911b). L' *Hæmogregarina canis* in Italia. *Ibid.*, XX, ii, 730.

—— (1912). Sull' *Anaplasma canis. Ibid.*. XXI, i, 231. *Pathologica*, IV, 358.

—— (1914). La meteorologia della leishmaniosi interna nel Mediterraneo. *Atti. R. Accad-Lincei. Rendie*, XXIII, i, 539.

—— (1914a). La meteorologia della leishmaniosi interna nel Mediterraneo. Contributo critico agli esperimenti di trasmissione. 2ª nota. *Ibid.*, XXIII. i, 625.

—— (1916). Leishmaniosi interna. *Annali d'Igiene*, XXVI, 248.

—— (1921). *Leishmania, Herpetomonas,* and *Crithidia* in fleas. *Parasitology*, XII, 366.

BASILE, C., & VISENTINI, A. (1911). Sull' identità delle leishmaniosi. (Culture su mezzo N.N.N. dei parassiti della leishmaniosi nel cane.) *Atti R. Accad. Lincei, Rendic.*, XX, i, 590.

BASS, C. C. (1914). Cultivation of malarial plasmodia *in vitro. Amer. Journ. Trop. Dis. & Prev. Med.*, I, 546.

—— (1922). Studies on inoculation of experimental animals with malaria. *Amer. Journ. Trop. Med.*, II, 107.

BASS, C. C., & JOHNS, F. M. (1912). The cultivation of malarial plasmodia (*Plasmodium vivax* and *Plasmodium falciparum*) *in vitro. Journ. Exp. Med.*, XVI, 567.

—— —— (1915). Pyorrhea dentalis and alveolaris. Specific cause and treatment. *Journ. Amer. Med. Assoc.*, LXIV, 553.

BASSETT-SMITH, P. W. (1919). The infection of their young by trypanosome-infected mothers. *Journ. Trop. Med. & Hyg.*, XXII, 198.

—— (1921). [Infection of fœtus of guinea-pig with trypanosomes.] *Trans. Roy. Soc. Trop. Med. & Hyg.*, XIV, 73.

BASTIANELLI, G., & BIGNAMI, A. (1899). (I.) Sulla struttura dei parassiti malarici e in specie dei gameti dei parassiti estivo-autunnali. (II.) Sullo sviluppo dei parassiti terzana nell' *Anopheles claviger. Annali d'Igiene*, IX, 245, 272.

—— —— (1900). Malaria and mosquitoes, a paper read before the tenth congress of the Società Italiana di Medicina Interna. (Translated by Dr. G. Sandison Brock.) *Lancet*, I. 79.

BASTIANELLI, G., BIGNAMI, A., & GRASSI, B. (1898). Coltivazione delle semilune malariche dell' uomo nell' *Anopheles claviger* Fabr. (sinonimo *Anopheles maculipennis* Meig.). Nota preliminare. *Atti R. Accad. Lincei, Rendic.*, VII, ii, 313. *Annali d'Igiene*, IX, 258.

BATTAGLIA, M. (1904). Alcune ricerche sopra due Tripanosomi (*Trypanosoma vespertilionis, Trypanosoma lewisi*). *Ann. Med. Nav.*, X, ii, 517.

—— (1914). Biologische Differentialcharaktere für einige Trypanosomen. *Centralbl. Bakt.*, I. Abt., LXXIV, 582.

BAUCHE, J., & MOTAIS, F. (1920). Sur un nouveau cas de dysenterie amibienne du chien. *Bull. Soc. Path. Exot.*, XIII, 161.

BAYON, H. (1915). Herpetomonidæ found in *Scatophaga hottentota* and *Chamæleon pumilus*. *Trans. Roy. Soc. S. Africa*, V, 61.

BEAUCHAMP, P. de (1911). *Astasia captiva* n. sp. Euglénien parasite de *Catenula lemnæ* Ant. Dug. *Arch. Zool. Exp.*, VI, N. & R., lii.

BECK, M. (1914). Untersuchungen über ein am Rovuma (Deutsch-Ostafrika) vorkommendes Trypanosoma beim Menschen. *Arch. Schiffs.- u. Trop.-Hyg.*, XVIII, 97.

BECK & WECK (1913). Die menschliche Trypanosomenkrankheit am Rovuma in Deutsch-Ostafrika. *Ibid.*, XVII, 145.

BECKER, E. R. (1923). Life-history studies of *Crithidia gerridis* in the water-strider. *Amer. Journ. Hyg.*, III, 202.

—— (1923a). Studies on the relationship between insect flagellates and leishmania. *Ibid.*, III, 462.

—— (1923b). Observations on the morphology and life-cycle of *Crithidia gerridis* Patton in the water-strider, *Gerris remigis* Say. *Journ. Parasit.*, IX. 141.

—— (1923c). Observations on the morphology and life-history of *Herpetomonas muscæ-domesticæ* in North American muscoid flies. *Ibid.*, IX, 199.

—— (1923d). Transmission experiments on the specificity of *Herpetomonas muscæ-domesticæ* in muscoid flies. *Ibid.*, X, 25.

—— (1925). The morphology of *Mastigina hylæ* (Frenzel) from the intestine of the tadpole. *Ibid.*, XI, 213.

BECKWITH, T. D., & REICH, W. W. (1921). The variation in the size of *Trypanosoma brucei* according to the host. *Proc. Soc. Exp. Biol. & Med.*, XVIII, 316.

BÉDIER, E. (1924). Piroplasme de la mangouste d'Afrique, *Herpestes calera* Erxleben. *C. R. Soc. Biol.*, XC, 415.

BEHN, P. (1910). Ueber Entwicklungsformen des *Trypanosoma franki*. *Berlin. Tierärztl. Woch.*, XXVI, 809.

—— (1910a). Infektion eines Kalbes mit Trypanosomen vom Typus des *Trypanosoma theileri* mittelst Blut von Kühen, in denen nur kulturell Flagellaten nachweisbar waren. *Ibid.*, XXVI, 998.

—— (1911). Trypanosomen beim Schafe. *Ibid.*, XXVII, 768.

—— (1912). Weitere Trypanosomenbefunde beim Schafe. *Ibid.*, XXVIII, 934.

BEHREND, K. (1914). Kurze Angaben über eine nichtpathogene Amöbe aus dem Darm von *Macacus rhesus*. *Arch. Protist.*, XXXIV, 35.

BĚLAŘ, K. (1915). Protozoenstudien. I. *Ibid.*, XXXVI, 13.

—— (1916). Protozoenstudien, II. *Ibid.*, XXXVI, 241.

—— (1920). Die Kernteilung von *Prowazekia*. *Ibid.*, XLI, 308.

—— (1921). Untersuchungen über Thecamöben der *Chlamydophrys*-Gruppe. *Ibid.*, XLIII, 287.

—— (1921a). Protozoenstudien, III. *Ibid.*, XLIII, 431.

—— (1921b). Morphologische und experimentelle Untersuchungen über die Befruchtung von *Actinophrys sol*. *Verhandl. Deutsch. Zool. Gesellsch.*, V, 46.

—— (1923). Untersuchungen an *Actinophrys sol* Ehrenberg. I. Die Morphologie des Formwechsels. *Arch. Protist.*, XLVI, 1.

—— (1924). Untersuchungen an *Actinophrys sol* Ehrenberg. II. Beiträge zur Physiologie des Formwechsels. *Ibid.*, XLVIII, 371.

—— (1926). Zur Cytologie von *Aggregata eberthi*. *Ibid.*, LIII, 312.

BELOU, P. (1903). *Tratado de Parasitologia Animal*. 1st edition, Buenos Aires, p. 62.

BENEDEN, P. J. VAN (1875). *Les commensaux et les parasites dans le règne animal*. Paris.

BEN-HAREL, S. (1923). Studies on bird malaria in relation to the mechanism of relapse. *Amer. Journ. Hyg.*, III, 652.

BENOIT-BAZILLE, H. (1920). Note sur une *Grahamella : Grahamella musculi*, n. sp., trouvée dans le sang de *Mus musculus*. *Bull. Soc. Path. Exot.*, XIII, 408.

BENSEN, W. (1908). Bau und Arten der Gattung *Lamblia*. *Zeitschr. Hyg. u. Infekt.*, LXI, 109.

—— (1909). Untersuchungen über *Trichomonas intestinalis* und *vaginalis* des Menschen. *Arch. Protist.*, XVIII, 115.

BENTLEY, C. A. (1904). A short note on the parasite of kala-azar. *Indian Med. Gaz.*, XXXIX, 81.

—— (1905). Preliminary note upon a leucocytozoon of the dog. *Brit. Med. Journ.*, I, 988.

—— (1911). *Nyssomyzomyia rossi* and malaria. *Paludism, being the Transactions of the Committee for the Study of Malaria in India*, No. 2.

BERENBERG-GOSSLER, H. (1909). Beiträge zur Naturgeschichte der Malariaplasmodien. *Arch. Protist.*, XVI, 245.

BERLINER, E. (1909). Flagellaten-Studien. *Ibid.*, XV, 297.

BERTRAM, A. (1892). Beiträge zur Kenntnis der Sarkosporidien. *Zool. Jahrb.*, Abt. Anat., V, 581.

BESNOIT, C., & ROBIN, V. (1912). Sarcosporidiose cutanée chez une vache. *Rev. Vét.*, XXXVII, 649.

BETTENCOURT, A., & BORGES, I. (1909). Sur une Theileria parasite du *Cephalophus grimmi* (L.). *Arq. R. Inst. Bact., Camara Pestana*, III, 19.

BETTENCOURT, A., & FRANÇA, C. (1905). Sur un trypanosome de la chauve-souris. *C. R. Soc. Biol.*, LIX, 305.

—— —— (1906). Sur un trypanosome du blaireau (*Meles taxus* Schr.). *Arq. R. Inst. Bact., Camara Pestana*, I, 73.

BETTENCOURT, A., FRANÇA, C., & BORGES, I. (1907). Un cas de piroplasmose bacilliforme chez le daim. *Ibid.*, I, 341.

BEVAN, LL. E. W. (1912). Anaplasmosis of sheep. *Vet. Journ.*, LXVIII, 400.

BEZZENBERGER, E. (1904). Ueber Infusorien aus asiatischen Anuren. *Arch. Protist.*, III, 138.

BIJLSMA, U. G. (1919). *Onderzoek naar het voorkomen van darmprotozoen bij Nederlanders*. Utrecht. (Review in *Trop. Dis. Bull.*, XIX, 718.)

BILLET, A. (1904). Culture d'un trypanosome de la grenouille chez une hirudinée; relation ontogénique possible de ce trypanosome avec une hémogrégarine. *C. R. Acad. Sci.*, CXXXIX, 574.

BISCHOFF, T. (1855). Über Ei- und Samenbildung und Befruchtung bei *Ascaris mystax*. *Zeitschr. Wiss. Zool.*, VI, 377.

BISHOP, C. F. (1912). Notes on a trypanosome found in a sheep tick, and its probable connection with the disease known as louping-ill. *Rept. 81st Meeting Brit. Assoc., Sec. D*, 418.

BITTER, (1905). [Texas fever in Egypt.] *Proc. 8th Internat. Congress Vet. Med.*, Budapest, III, 289.

BIZARD, E., & TERRIEN, E. (1923). Cas de leishmaniose interne chez une adulte contracté en France. *Bull. Soc. Path. Exot.*, XVI, 89.

BLACKLOCK, B. (1912). The trypanosomes found in a horse naturally infected in the Gambia. *Ann. Trop. Med. & Parasit.*, VI, 107.

—— (1914). On the multiplication and infectivity of *T. cruzi* in *Cimex lectularius*. *Brit. Med. Journ.*, I, 912.

—— (1923). The aetiology of kala-azar and tropical sore. *Lancet*, II, 273.

BLACKLOCK, B., & ADLER, S. (1922). A parasite resembling *Plasmodium falciparum* in a chimpanzee. *Ann. Trop. Med. & Parasit.*, XVI, 99.

—— —— (1924). A malaria parasite of the chimpanzee. *Ibid.*, XVIII, 1.

BLACKLOCK, B., & YORKE, W. (1913). The trypanosomes causing dourine (mal de coït or Beschäl-seuche). *Proc. Roy. Soc.*, B, LXXXVII, 89.

—— —— (1913a). *Trypanosoma vivax* in rabbits. *Ann. Trop. Med. & Parasit.*, VII, 563.

BLANC, G. (1917). Sur un cas de toxoplasmose canine observé en Tunisie. *Bull. Soc. Path. Exot.*, X, 377.

BLANCHARD, M., & LEFROU, G. (1922). Présence de spirochètes dans le sang d'européens atteints de fièvre bilieuse hémoglobinurique. Le problème étiologique de cette spirochétose. *Ibid.*, XV, 699.

BLANCHARD, R. (1885). Note sur les Sarcosporidies et sur un essai de classification de ces Sporo-zoaires. *Bull. Soc. Zool.*, Paris, X, 244.

—— (1888). Etude sur une maladie spéciale des mulets importés au Tonkin pour le service de l'armée et considérations sur l'acclimatement de l'espèce chevaline en Extrême Orient. *Bull. Soc. Centr. Méd. Vét.*, XLII, 694.

—— (1888a). Remarques sur le mégastome intestinal. *Bull. Soc. Zool.*, Paris, XIII, 18.

—— (1900). Les coccidies et leur rôle pathogène. *Caus. Sci. Soc. Zool.*, Paris, No. 5, 133.

—— (1904). Sur un travail de M. le Dr. Brumpt intitulé: Quelques faits relatifs à la transmission de la maladie du sommeil par les mouches tsétsé. *Arch. Parasit.*, VIII, 573.

BLANCHARD, R., & BLATIN, M. (1907). Immunité de la marmotte en hibernation à l'égard des maladies parasitaires. *Ibid.*, XI, 361.

BLANCHARD, R., & LANGERON, M. (1913). Le paludisme des macaques (*Plasmodium cynomolgi* Mayer, 1907). *Ibid.*, XV, 529.

—— —— (1913a). Nouvelles recherches sur le paludisme des macaques, d'après les notes posthumes de Xavier Bouniol. *Ibid.*, XV, 599.

BLOCHMANN, F. (1884). Bemerkungen über einige Flagellaten. *Zeitschr. Wiss. Zool.*, XL, 42.

—— (1886). Über eine neue Hæmatococcus-Art. (Habilitationsschrift). Heidelberg.

—— (1886a). *Die Mikroskopische Pflanzen- und Thierwelt des Süsswassers*. Braunschweig.

—— (1894). Zur Kenntnis von *Dimorpha mutans* Grub. *Biol. Centralbl.*, XIV, 197.

—— (1895). Protozoa. Kirchner u. Blochmann's *Die mikroskopische Pflanzen- und Thierwelt des Süsswassers*. Theil 2. Abt. I, 2nd edition, Hamburg.

BOECK, W. C. (1917). Mitosis in *Giardia microti*. *Univ. California Publ. Zool.*, XVIII, 1.

—— (1917a). A rapid method of the detection of Protozoan cysts in mammalian fæces. *Ibid.*, XVIII, 145.

—— (1919). Studies on *Giardia microti*. *Ibid.*, XIX, 85.

—— (1921). A Protozoan survey of an industrial school for boys and girls. *Journ. Parasit.*, VII, 191.

BOECK, W. C. (1921a). *Chilomastix mesnili* and a method for its culture. *Journ. Exp. Med.*, XXXIII, 147.

—— (1921b). The thermal death-point of the human intestinal Protozoan cysts. *Amer. Journ. Hyg.*, I, 365.

—— (1921c). On the longevity of human intestinal Protozoan cysts. *Ibid.*, I, 527.

—— (1924). Studies on *Tricercomonas intestinalis* (Wenyon and O'Connor, 1917). *Amer. Journ. Trop. Med.*, IV, 519.

BOECK, W. C., & DEBOHLAV, J. (1925). The cultivation of *Endamœba histolytica*. *Proc. Nat. Acad. Sci.*, Washington, II, 235; *Amer. Journ. Hyg.*, V, 371.

BOECK, W. C., & STILES, C. W. (1923). Studies on various intestinal parasites (especially amœbæ) of man. Bull. No. 133, Hyg. Lab., Washington.

BOËZ, L. (1921). Schizogonie et lésions pulmonaires dans un cas de toxoplasmose spontanée du chien. *C. R. Soc. Biol.*, LXXXV, 479.

BOGOYAVLENSKY, N. (1922). *Menzbieria hydrachnœ* n. g., n. sp. *Arch. Soc. Russe Protist.*, I, 10.

BOHNE, A., & PROWAZEK, S. VON (1908). Zur Frage der Flagellatendysenterie. *Arch. Protist.*, XII, 1.

BONEY, T. K., CROSSMAN, L. G., & BOULENGER, C. L. (1918). Report of a base laboratory in Mesopotamia for 1916, with special reference to water-borne diseases. *Journ. R. Army Med. Corps*, XXX, 409.

BONOME, A. (1895). Über parasitäre Iktero-Hämaturie der Schafe. *Arch. Path. Anat.*, CXXXIX, 1.

BOON VAN OSTADE, C. M. (1923). Coccidiosis bij den mensch. *Geneesk. Tijdschr. Nederl.-Indie*, LXIII, 670.

BOSKAMP, E. (1922). Ueber Bau, Lebensweise und systematische Stellung von *Selenomonas palpitans* (Simons). *Centralbl. Bakt.*, I. Abt., LXXXVIII, 58.

BOTT, K. (1906). Ueber die Fortpflanzung von *Pelomyxa palustris* nebst Mittheilung über ihren Bau. *Arch. Protist.*, VIII, 120.

BOUET, G. (1907). Les trypanosomes animales de la Basse-Côte d'Ivoire. *Ann. Inst. Pasteur*, XXI, 468.

—— (1909). Sur deux hémocytozoaires pigmentés des reptiles. *C. R. Soc. Biol.*, LXVI, 43.

—— (1909a). Sur quelques trypanosomes des vertébrés à sang froid de l'Afrique occidentale française. *Ibid.*, LXVI, 609.

BOUET, G., & ROUBAUD, E. (1910). Expériences diverses de transmission des trypanosomes par les glossines. I-II. *Ann. Inst. Pasteur*, XXIV, 658; III. *Bull. Soc. Path. Exot.*, III, 599; IV. *ibid.*, 722.

—— —— (1911). Sur la présence au Dahomey et le mode de transmission du *Leptomonas davidi* Lafont, flagellé parasite des Euphorbiacées. *C. R. Soc. Biol.*, LXX, 55.

—— —— (1911a). Expériences diverses de transmission des trypanosomes par les glossines. V. Transmission naturelle de la souma (*T. cazalboui*) par *Glossina tachinoides* et *morsitans* ; de la baléri (*T. pecaudi*) par *Glossina morsitans*, au Soudan Nigérien, en saison sèche. *Bull. Soc. Path. Exot.*, IV, 539.

—— —— (1912). Expériences diverses de transmission des trypanosomes par les glossines. VI. Trypanosomiases et glossines de la Haute-Gambie et de la Casamance. Expériences diverses de transmission par *Glossina palpalis* et *morsitans*. *Ibid.*, V, 204.

—— —— (1912a). Expériences de transmission des trypanosomiases animales de l'Afrique occidentale française, par les stomoxes. *Ibid.*, V, 544.

—— —— (1912b). La piroplasmose (nuttalliose) de l'âne en Afrique occidentale. *Ibid.*, V, 806.

BOUFFARD, G. (1907). Sur l'étiologie de la souma, trypanosomiase du Soudan français. *C. R. Soc. Biol.*, LXII, 71.

—— (1909). Le rôle enzootique de la *Glossina palpalis* dans la souma. *Bull. Soc. Path. Exot.*, II, 599.

—— (1910). *Glossina palpalis* et *Trypanosoma cazalboui*. *Ann. Inst. Pasteur*, XXIV, 276.

BOUILLIEZ, M. (1913). Nouvelles recherches expérimentales sur un Plasmodium des singes. *C. R. Soc. Biol.*, LXXIV, 1070.

—— (1916). Un cas de kala-azar infantile au Moyen-Chari (Territoire du Tchad). *Bull. Soc. Path. Exot.*, IX, 299.

—— (1917). Auto-observation d'un cas d'inoculation accidentelle de bouton d'orient sur la conjonctive. *Ibid.*, X, 1.

—— (1917a). Recherches expérimentales sur *Leishmania tropica*. *Ibid.*, X, 66.

BOURRET, G. (1911). La toxoplasmose du lapin à Saint-Louis du Sénégal. *Ibid.*, IV, 373.

BOUSFIELD, L. (1911). Remarks on kala-azar in the Kassala and Blue Nile districts of the Sudan. *4th Rept., Wellcome Trop. Res. Labs.*, Khartoum, A, 127.

BOWHILL, T. (1906). Note on Hæmatozoa observed in a bat, and the occurrence of *Acanthia pipistrelli* Jenyns in South Africa. *Journ. Hyg.*, VI, 246.

—— (1909). Note on the occurrence of a large flagellate associated with piroplasmata infection in a cow in British Columbia. *Journ. Trop. Vet. Sci.*, IV, 104.

BOWMAN, F. B. (1909). Two cases of *Balantidium coli* infection, with autopsy. *Philippine Journ. Sci.* (*B. Med. Sci.*), IV, 417.

—— (1911). A case of dysentery caused by *Balantidium coli*, with coincident filarial infarction of the spleen. *Ibid.*, VI, 147.

BOYD, M. F. (1918). A note on the cultivation of *Trichomonas intestinalis*. *Journ. Parasit.*, IV, 168.

—— (1919). Observations upon *Trichomonas intestinalis in vitro*. *Ibid.*, V, 132.

BOYD, W. (1921). The detection of *Lamblia* (*Giardia*) *intestinalis* by means of the duodenal tube. *Canadian Med. Assoc. Journ.*, XI, 658.

BOYERS, L. M., KOFOID, C. A., & SWEZY, O. (1925). Chronic human amebiasis. *Journ. Amer. Med. Assoc.*, LXXXV, 1441.

BRAHMACHARI, U. (1920). *Kala-azar and its treatment*. 2nd edition. Calcutta.

BRAHMACHARI, U. N. (1922). Chemotherapy of antimonial compounds in kala-azar infection. *Indian Journ. Med. Res.*, X, 492.

BRAMACHARI, U. N., & SEN, P. B. (1923). The globulin opacity test for kala-azar. *Indian Med. Gaz.*, LVIII, 295.

BRASIL, L. (1904). Sur une coccidie nouvelle, parasite d'un cirratulien. *C. R. Acad. Sci.* CXXXIX, 645.

—— (1905). Nouvelles recherches sur la reproduction des grégarines monocystidées. *Arch. Zool. Exp.*, IV, 69.

—— (1907). Recherches sur le cycle évolutif des Selenidiidæ, grégarines parasites d'annélides polychètes. I. La schizogonie et la croissance des gamétocytes chez *Selenidium caulleryi* n. sp. *Arch. Protist.*, VIII, 370.

—— (1909). Documents sur quelques sporozoaires d'annélides. *Ibid.*, XVI, 107.

BRAULT, J. (1898). Contribution à la géographie médicale des maladies africaines. *Janus*, III, 36.

BRAUN, M. (1908). *Die tierischen Parasiten des Menschen*. 4th edition. Würzburg.

BRAUNE, R. (1913). Untersuchungen über die im Wiederkäuermagen vorkommenden Protozoen. *Arch. Protist.*, XXXII, 111.

BREEST, F. (1914). Zur Kenntnis der Symbiontenübertragung bei viviparen Cocciden und bei Psylliden. *Ibid.*, XXXIV, 263.

BREINL, A. (1912). *Report for the year* 1911, *Australian Institute of Tropical Medicine*. Sydney.

BREINL, A., & HINDLE, E. (1908). Contributions to the morphology and life-history of *Piroplasma canis*. *Ann. Trop. Med. & Parasit.*, II, 233.

BRESSLAU, E., & BUSCHKIEL, M. (1919). Die Parasiten der Stechmückenlarven. *Biol. Centralbl.*, XXXIX, 325.

BRIMONT, E. (1909). Sur quelques hématozoaires de la Guyane. Première note. *C. R. Soc. Biol.*, LXVII, 169.

BRODEN, A. (1904). Les infections à trypanosomes au Congo chez l'homme et les animaux. *Bull. Soc. Etudes Colon.*, February.

BRODEN, A., & RODHAIN, J. (1908). Le liquide cérébro-spinal dans la trypanosomiase humaine. *Bull. Soc. Path. Exot.*, I, 496.

—— (1910). Action de l'émétique sur le *Trypanosoma angolense* (*s. cazalboui*). *Ibid.*, III, 233.

BROOKS, H. (1903). A few animal parasites sometimes found in man. *Proc. New York Path. Soc.* (N.S.), III, 28.

BROUGHTON-ALCOCK, W. (1921). Laboratory observations on pensioners who contracted malaria in the late war. · *Proc. Roy. Soc. Med.*, XIV (Sect. Trop. Dis. & Parasit.), 45.

BROUGHTON-ALCOCK, W., & THOMSON, J. G. (1922). *Eimeria oxyspora*, Dobell, 1919, found in a specimen of human fæces in England. *Ibid.*, XV (Sect. Trop. Dis. & Parasit.), 1.

—— (1922a). *Embadomonas intestinalis* (Wenyon and O'Connor), 1917; description of the cysts and free forms found in a case in England. *Ibid.*, XV (Sect. Trop. Dis. & Parasit.), 8.

BROWN H. C. (1922). Observations on the amœbicidal action of conessine. *Brit. Med. Journ.*, I, 993.

BROWN, H. C., & STAMMERS, G. E. F. (1922). Observations on canine fæces on London pavements. *Lancet*, II, 1165.

BRUCE, D. (1895). *Preliminary report on the tsetse fly disease or nagana in Zululand*. Umbobo, December, 1895.

—— (1897). *Further report on the tsetse fly disease or nagana in Zululand*. Umbobo, May 29, 1896. London.

—— (1903). *Appendix to further report on the tsetse fly disease or nagana in Zululand*. London.

—— (1911). The morphology of *Trypanosoma evansi* (Steel). *Proc. Roy. Soc.*, B, LXXXIV, 181; *Rep. Sleep. Sickn. Comm. Roy. Soc.*, XII, 19.

—— (1914). Classification of the African trypanosomes pathogenic to man and domestic animals. *Trans. Soc. Trop. Med. & Hyg.*, VIII, 1.

BRUCE, D., & BATEMAN, H. R. (1908). Have trypanosomes an ultramicroscopic stage in their life-history ? *Proc. Roy. Soc.* B, LXXX, 394.

BRUCE, D., HAMERTON, A. E., & BATEMAN, H. R. (1911). Experiments to ascertain if the domestic fowl of Uganda may act as a reservoir of the virus of sleeping sickness (*Trypanosoma gambiense*). *Proc. Roy. Soc.*, B, LXXXIII, 328; *Rep. Sleep. Sickn. Comm .Roy. Soc.*, XI, 94.

BRUCE, D., HAMERTON, A. E., BATEMAN, H. R., & MACKIE, F. P. (1909). The development of *Trypanosoma gambiense* in *Glossina palpalis*. *Proc. Roy. Soc.*, B, LXXXI, 405; *Rep. Sleep. Sickn. Comm. Roy. Soc.*, X, 46.

—————————— (1909a). *Trypanosoma ingens*, n. sp. *Proc. Roy. Soc.*, B, LXXXI, 323; *Rep. Sleep. Sickn. Comm. Roy. Soc.*, X, 27.

—————————— (1909b). A note on the occurrence of a trypanosome in the African elephant. *Proc. Roy. Soc.*, B, LXXXI, 414; *Rep. Sleep. Sickn. Comm. Roy. Soc.*, X, 54.

—————————— (1910). Sleeping sickness in Uganda. Duration of the infectivity of the *Glossina palpalis* after the removal of the lake-shore population. *Proc. Roy. Soc.*, B, LXXXII, 56; *Rep. Sleep. Sickn. Comm. Roy. Soc.*, X, 56.

—————————— (1910a). The development of trypanosomes in tsetse flies. *Proc. Roy. Soc.*, B, LXXXII, 368; *Rep. Sleep. Sickn. Comm. Roy. Soc.*, XI, 12.

—————————— (1910b). Trypanosome diseases of domestic animals in Uganda. I. *Trypanosoma pecorum*. *Proc. Roy. Soc.*, B, LXXXII, 468; *Rep. Sleep. Sickn. Comm. Roy. Soc.*, XI, 128.

—————————— (1910c). Experiments to ascertain if cattle may act as a reservoir of the virus of sleeping sickness (*Trypanosoma gambiense*). *Proc. Roy. Soc.*, B, LXXXII, 480; *Rep. Sleep. Sickn. Comm. Roy. Soc.*, XI, 71.

—————————— (1910d). Trypanosome diseases of domestic animals in Uganda. II. *Trypanosoma brucei* (Plimmer and Bradford). *Proc. Roy. Soc.*, B, LXXXIII, 1; *Rep. Sleep. Sickn. Comm. Roy. Soc.*, XI, 140.

—————————— (1910e). Trypanosome diseases of domestic animals in Uganda. III. *Trypanosoma vivax* (Ziemann). *Proc. Roy. Soc.*, B, LXXXIII, 15; *Rep. Sleep. Sickn. Comm. Roy. Soc.*, XI, 150.

—————————— (1910f). Mechanical transmission of sleeping sickness by the tsetse fly. *Proc. Roy. Soc.*, B, LXXXII, 498; *Rep. Sleep. Sickn. Comm. Roy. Soc.*, XI, 66.

—————————— (1911a). Trypanosome diseases of domestic animals in Uganda. IV. *Trypanosoma uniforme* sp. nov. *Proc. Roy. Soc.*, B, LXXXIII, 176; *Rep. Sleep. Sickn. Comm. Roy. Soc.*, XI, 160.

—————————— (1911b). Trypanosome diseases of domestic animals in Uganda. V. *Trypanosoma nanum*, Laveran. *Proc. Roy. Soc.*, B, LXXXIII, 180; *Rep. Sleep. Sickn. Comm. Roy. Soc.*, XI, 164.

—————————— (1911c). Experiments to ascertain if *Trypanosoma gambiense* during its development within *Glossina palpalis* is infective. *Proc. Roy. Soc.*, B, LXXXIII, 345.

—————————— (1911d). Further researches on the development of *Trypanosoma gambiense* in *Glossina palpalis*. *Proc. Roy. Soc.*, B, LXXXIII, 513; *Rep. Sleep. Sickn. Comm. Roy. Soc.*, XI, 34.

BRUCE, D., HAMERTON, A. E., BATEMAN, H. R., MACKIE, F. P., & LADY BRUCE (1911e). Experiments to ascertain whether the mammals, birds or reptiles in Uganda, living on or near the lake-shore, harbour *Trypanosoma gambiense*. *Rep. Sleep. Sickn. Comm. Roy. Soc.*, XI, 100.

—————————— (1911f). A trypanosome found in the blood of a crocodile. *Ibid.*, XI, 184.

—————————— (1911g). The occurrence of a piroplasmosis in a white mouse. *Ibid.*, XI, 189.

—————————— (1911h). Further researches on the development of *Trypanosoma vivax* in laboratory-bred *Glossina palpalis*. *Ibid.*, XI, 50.

—————————— (1911i). *Trypanosoma gallinarum* n. sp. *Ibid.*, XI, 170.

—————————— (1911j). Experiments to ascertain if there is an ultra-microscopical phase in the development of *Trypanosoma gambiense* in *Glossina palpalis*. *Ibid.*, XI, 122.

BRUCE, D., HAMERTON, A. E., WATSON, D. P., & LADY BRUCE (1914). Description of a strain of *Trypanosoma brucei* from Zululand. Pt. I. Morphology. *Proc. Roy. Soc.*, B, LXXXVI, 493; *Rep. Sleep. Sickn. Comm. Roy. Soc.*, XVI, 71.

—————————— (1914a). The trypanosome causing disease in man in Nyasaland. Pt. III. Development in *Glossina morsitans*. *Proc. Roy. Soc.*, B, LXXXVII, 516; *Rep. Sleep. Sickn. Comm. Roy. Soc.*, XVI, 135.

—————————— (1914b). *Glossina brevipalpis* as a carrier of trypanosome disease in Nyasaland. *Proc. Roy. Soc.*, B, LXXXVIII, 20; *Rep. Sleep. Sickn. Comm. Roy. Soc.*, XVI, 25, 157, 161, 179, 187.

—————————— (1914c). Trypanosome diseases of domestic animals in Nyasaland. III. *Trypanosoma pecorum*. Development in *Glossina morsitans*. *Proc. Roy. Soc.*, B, LXXXVIII, 33; *Rep. Sleep. Sickn. Comm. Roy. Soc.*, XVI, 175.

—————————— (1914d). Trypanosomes found in wild *Glossina morsitans* and wild game in the fly belt of the Upper Shiré Valley. *Proc. Roy. Soc.*, B, LXXXVIII, 38; *Rep. Sleep. Sickn. Comm. Roy. Soc.*, XVI, 21.

BRUCE, D., HAMERTON, A. E., WATSON, D. P., & LADY BRUCE (1914e). Trypanosome diseases of domestic animals in Nyasaland. *Trypanosoma capræ* (Kleine). Pt. III. Development in *Glossina morsitans*. *Proc. Roy. Soc.*, B, LXXXVIII, 92; *Rep. Sleep. Sickn. Comm. Roy. Soc.*, XVI. 183.

————————— (1914f). Infectivity of *Glossina morsitans* in Nyasaland during 1912 and 1913. *Proc. Roy. Soc.*, B, LXXXVIII, 43; *Rep. Sleep. Sickn. Comm. Roy. Soc.*, XVI, 15.

————————— (1914g). Morphology of various strains of the trypanosome—*Trypanosoma brucei* vel *rhodesiense*—causing disease in man in Nyasaland. The naturally infected dog strain. *Proc. Roy. Soc.*, B, LXXXVIII, 111 & 130; *Rep. Sleep. Sickn. Comm. Roy. Soc.*, XVI, 88.

————————— (1914h). Description of a strain of *Trypanosoma brucei* from Zululand. Pt. III. Development in *Glossina morsitans*. *Proc. Roy. Soc.*, B, LXXXVII, 526; *Rep. Sleep. Sickn. Comm. Roy. Soc.*, XVI, 145.

————————— (1914i). The trypanosome causing disease in man in Nyasaland. The naturally infected dog strain. Pt. III. Development in *Glossina morsitans*. *Proc. Roy. Soc.*, B, LXXXVIII, 213; *Rep. Sleep. Sickn. Comm. Roy. Soc.*, XVI, 151.

BRUCE, D., HARVEY, D., HAMERTON, A. E., & LADY BRUCE (1913). Morphology of various strains of the trypanosome causing disease in man in Nyasaland. The wild *Glossina morsitans* strain. *Proc. Roy. Soc.*, B, LXXXVI, 408; *Rep. Sleep. Sickn. Comm. Roy. Soc.*, XV, 61.

————————— (1913a). Infectivity of *Glossina morsitans* in Nyasaland. *Proc. Roy. Soc.*, B, LXXXVI, 422; *Rep. Sleep. Sickn. Comm. Roy. Soc.*, XIV, 73.

————————— (1913b). Trypanosome diseases of domestic animals in Nyasaland. III. *Trypanosoma pecorum*. *Proc. Roy. Soc.*, B, LXXXVII, 1; *Rep. Sleep. Sickn. Comm. Roy. Soc.*, XV, 78.

————————— (1913c). *Plasmodium cephalophi* sp. nov. *Proc. Roy. Soc.*, B, LXXXVII, 45; *Rep. Sleep. Sickn. Comm. Roy. Soc.*, XVI, 203.

————————— (1913d). Trypanosomes of the domestic animals in Nyasaland. I. *Trypanosoma simiæ* sp. nov. Part II.—The susceptibility of various animals to *T. simiæ*. Part III. *Proc. Roy. Soc.*, B, LXXXVII, 48, 58; *Rep. Sleep. Sickn. Comm. Roy. Soc.*, XV, 121.

BRUCE, D., HARVEY, D., HAMERTON, A. E., DAVEY, J. B., & LADY BRUCE (1912). The morphology of *Trypanosoma simiæ*. *Proc. Roy. Soc.*, B, LXXXV, 477; *Rep. Sleep. Sickn. Comm. Roy. Soc.*, XV, 11.

————————— (1913e). The trypanosomes found in the blood of wild animals living in the sleeping-sickness area, Nyasaland. *Proc. Roy. Soc.*, B, LXXXVI, 269; *Rep. Sleep. Sickn. Comm. Roy. Soc.*, XV, 16.

————————— (1913f). Trypanosome diseases of domestic animals in Nyasaland. II. *Trypanosoma capræ* (Kleine). *Proc. Roy. Soc.*, B, LXXXVI, 278; *Rep. Sleep. Sickn. Comm. Roy. Soc.*, XV, 25.

————————— (1915). The development in *Glossina morsitans* of *Trypanosoma gambiense*, Tanganyika. *Rep. Sleep. Sickn. Comm. Roy. Soc.*, XVI, 201.

————————— (1915a). Trypanosomes and other parasites of animals in Nyasaland. *Ibid.*, XVI, 203.

BRUCE, D., & NABARRO, D. (1903). Progress report on sleeping sickness in Uganda. *Ibid.*, I, 11.

BRUCE, D., NABARRO, D., & GREIG, E. D. W. (1903). Can the Uganda tsetse fly (*Glossina palpalis*) carry the trypanosome found in sleeping-sickness cases to healthy animals ? *Ibid.*, IV. 56.

BRUCE, E. A. (1919). A preliminary note on a new coccidium of rabbits. *Journ. Amer. Vet. Med. Assoc.*, LV, 620.

BRUG, S. L. (1916). Morphologische Studien an *Proteosoma præcox*. *Arch. Schiffs.- u. Trop.-Hyg.*, XX, 289.

——(1917). Een paar nieuw ontdekte darmparasieten. *Geneesk. Tijdschr. Nederl.-Indie*, LVII, 570.

——(1918). Enkele opmerkingen over de nomenclatuur van eenige nieuwere parasieten uit den menschelijken darm. *Ibid.*, LVIII, 283.

——(1918a). *Entamœba cuniculi* n. sp. *Ibid.*, LVIII, 811.

——(1919). *Endolimax williamsi* : the amœboid form of the iodine cysts. *Indian Journ. Med. Res.*, VI, 386.

——(1919a). De entamœben van de rat. *Jaarverslag Cent. Milit. Geneesk. Lab.*, 1918, Batavia, 127.

——(1920). Onderzoek naar het voorkomen van dierlijke darmparasieten bij nietbuiklijders, *Geneesk. Tijdschr. Nederl.-Indie*, LX, 626.

——(1920a). *Endolimax kueneni* n. sp. parasitic in the intestinal tract of the monkey, *Macacus cynomolgus*. *Parasitology*, XII, 378.

——(1921). Die Iodzysten. *Arch. Schiffs- u. Trop.,-Hyg.*, XXV, 47.

——(1922). Quelques observations sur les protozoaires, parasites intestinaux de l'homme et des animaux. *Bull. Soc. Path. Exot.*, XV, 132.

BRUG. S. L. (1922a). Coccidiose bij den mensch. *Geneesk. Tijdschr. Nederl.-Indië*, LXII, 363.
—— (1923). Protozooögische waarnemingen. *Ibid.*, LXIII. 620.
BRUG, S. L., DEN HEYER, J. K., & HAGA, J. (1925). Toxoplasmose du lapin aux Indes orientales néerlandaises. *Ann. Parasit. Hum. & Comp.*, III, 232.
BRUGNATELLI, E. (1908). Observations sur une Coccidie (*Klossiella muris*) ayant donné occasion à une nouvelle doctrine sur la fonction rénale. *Journ. Anat. & Physiol.*, Paris, LXIV, 121.
BRUMPT, E. (1901). Notes et observations sur les maladies parasitaires. *Arch. Parasit.*, IV, 563.
—— (1904). Contribution à l'étude de l'évolution des hémogrégarines et des trypanosomes. *C. R. Soc. Biol.*, LVII, 165.
—— (1905). Trypanosomes et trypanosomoses. *Rev. Scient.*, IV, 321.
—— (1905a). Au sujet du traitement de la maladie du sommeil, réponse à M. le Professeur Laveran. *C. R. Soc. Biol.*, LIX, 316.
—— (1906). Sur quelques espèces nouvelles de trypanosomes parasites des poissons d'eau douce; leur mode d'évolution. *Ibid.*, LX, 160.
—— (1906a). Mode de transmission et évolution des trypanosomes des poissons; description de quelques espèces de trypanoplasmes des poissons d'eau douce; trypanosome d'un crapaud africain. *Ibid.*, LX, 162.
—— (1906b). Expériences relatives au mode de transmission des trypanosomes et des trypanoplasmes par les hirudinées. *Ibid.*, LXI, 77.
—— (1906c). Rôle pathogène et mode de transmission du *Trypanosoma inopinatum* Ed. et Ét. Sergent. Mode d'inoculation d'autres trypanosomes. *Ibid.*, LXI, 167.
—— (1907). De l'hérédité des infections à trypanosomes et à trypanoplasmes chez les hôtes intermédiaires. *Ibid.*, LXIII, 176.
—— (1908). De l'origine des hémoflagellés du sang des vertébrés. *Ibid.*, LXIV, 1046.
—— (1908a). Guérison de la maladie du sommeil chez le lérot vulgaire en hibernation. Action du froid sur le *Trypanosoma inopinatum in vivo*. *Ibid.*, LXIV, 1147.
—— (1908b). Globules géants ou "corps en demi-lune" du paludisme; autres altérations globulaires au cours de cette maladie infectieuse. *Bull. Soc. Path. Exot.*, I, 201.
—— (1909). Démonstration du rôle pathogène du *Balantidium coli*, enkystement et conjugaison de cet infusoire. *C. R. Soc. Biol.*, LXVII, 103.
—— (1909a). [Remarks on entamoebæ and *Trichomonas* in monkeys.] *Bull. Soc. Path. Exot.*, II, 20.
—— (1909b). Sur un nouveau trypanosome non pathogène du singe. *Ibid.*, II, 267.
—— (1910). Sur quelques particularités morphologiques et physiologiques des trypanosomes (perte du flagelle et formation de pigments divers). *Ibid.*, III, 366.
—— (1910a). *Précis de parasitologie*. Paris.
—— (1911). Note sur le parasite des hématies de la taupe: *Grahamella talpæ* n. g., n. sp. *Bull. Soc. Path. Exot.*, IV, 514.
—— (1912). Le *Trypanosoma cruzi* évolue chez *Conorhinus megistus*, *Cimex lectularius*, *Cimex boueti* et *Ornithodorus moubata*. Cycle évolutif de ce parasite. *Ibid.*, V, 360.
—— (1912a). Côlite à *Tetramitus mesnili* (Wenyon, 1910) et côlite à *Trichomonas intestinalis* Leuckart, 1879. *Blastocystis hominis* n. sp. et formes voisines. *Ibid.*, V, 725.
—— (1913). Évolution de *Trypanosoma lewisi, duttoni, nabiasi, blanchardi*, chez les puces et les punaises. Transmission par les déjections. Comparaison avec *T. cruzi*. *Ibid.*, VI, 167.
—— (1913a). Immunité partielle dans les infections à *Trypanosoma cruzi*, transmission de ce trypanosome par *Cimex rotundatus*. Rôle régulateur des hôtes intermédiaires. Passage à travers la peau. *Ibid.*, VI, 172.
—— (1913b). A propos de *l'Hæmocystozoon brasiliense* de Franchini. *Ibid.*, VI, 377.
—— (1913c). *Précis de parasitologie*. 2nd edition. Paris.
—— (1913d). Études sur les infusoires parasites. I. La conjugaison d'*Anoplophrya circulans* Balbiani, 1885. *Arch. Parasit.*. XVI, 187.
—— (1914). Importance du cannibalisme et de la coprophagie chez les reduvidés hématophages (*Rhodnius, Triatoma*) pour la conservation des trypanosomes pathogènes en dehors de l'hôte vertébré. *Bull. Soc. Path. Exot.*, VII, 702.
—— (1914a). Le xénodiagnostic. Application au diagnostic de quelques infections parasitaires et en particulier à la trypanosomose de Chagas. *Ibid.*, VII, 706.
—— (1915). Cycle évolutif des Opalines. *Ibid.*, VIII, 397.
—— (1918). Protozoaires et helminthes des selles aux armées. *C. R. Soc. Biol.*, LXXXI, 1044.
—— (1919). Transmission de la piroplasmose canine française par le *Dermacentor reticulatus*. Embolies parasitaires dans les capillaires de l'encéphale. *Bull. Soc. Path. Exot.*, XII, 651.
—— (1919a). Transmission de la piroplasmose canine tunisienne par le *Rhipicephalus sanguineus*. *Ibid.*, XII, 757.
—— (1920). Les piroplasmes des bovidés et leurs hôtes vecteurs. *Ibid.*, XIII, 416.
—— (1922). *Précis de parasitologie*. 3rd edition. Paris.
—— (1923). Les theilerioses mortelles du Bassin Méditerranéen sont dues à *Theileria mutans*. *Ann. Parasit. Hum. & Comp.*, I, 16.

BRUMPT, E. (1924). Les piroplasmes des bovidés. I. Les theileries. *Ibid.*, II, 340.

—— (1925). Etude sommaire de l' *Entamœba dispar* n. sp. amibe à kystes quadrinucléés, parasite de l'homme. *Bull. Acad. Méd.*, XCIV, 943.

BRUMPT, E., & GOMES, J. F. (1914). Description d'une nouvelle espèce de *Triatoma* (*T. chagasi*) hôte primitif du *Trypanosoma cruzi* (Chagas). *Ann. Paulist. Med. & Cir.*, III, No. 4, p. 73.

BRUMPT, E., & GONZALEZ-LUGO (1913). Présentation d'un réduvide du Vénézuela, le *Rhodnius prolixus*, chez lequel évolue *Trypanosoma cruzi*. *Bull. Soc. Path. Exot.*, VI, 382.

BRUMPT, E., & JOYEUX, C. (1912). Sur un infusoire nouveau, parasite du chimpanzé, *Troglodytella abrassarti* n. g., n. sp. *Ibid.*, V, 499.

BRUMPT, E., & LARROUSSE, F. (1922). Transmission de la piroplasmose canine française par le *Dermacentor venustus*. *Ibid.*, XV, 540.

BRUMPT, E., & LAVIER, G. (1924). Un nouvel euglénien polyflagellé parasite du têtard de *Leptodactyllus ocellatus* au Brésil. *Ann. Parasit. Hum. & Comp.*, II, 248.

BRUMPT, E., & PEDROSO, A. (1913). Recherches épidémiologiques sur la leishmaniose forestière américaine dans l'Etat de Sâo-Paulo (Brésil). *Bull. Soc. Path. Exot.*, VI, 752; *Ann. Paulist. Med. & Cir.*, I. 97.

BRUMPT & WURTZ (1904). Essais de traitement de la maladie du sommeil expérimentale. (Note préliminaire.) *C. R. Soc. Biol.*, LVI, 756.

BUCHNER (1922). Sind die Crithidien der Schaflaus für Mäuse pathogen ? *Zeitschr. Hyg. u. Infekt.*, XCV, 115.

BÜTSCHLI, O. (1878). Beiträge zur Kenntnis der Flagellaten und einiger verwandten Organismen. *Zeitschr. Wiss. Zool.*, XXX, 205.

—— (1881). Myxosporidia. *Zool. Jahresber.* für 1880, I. Abt., 162.

—— (1882–1889). Protozoa. Bronn's *Kl. u. Ordn. d. Thier-Reichs.* Leipzig.

BUGGE & HEINKE (1921). Ueber das Vorkommen von Coccidien beim Meerschweinchen. *Deutsch. Tierärztl. Woch.*, XXIX, 41.

BUISSON, J. (1923). Les infusoires ciliés du tube digestif de l'homme et des mammifères. *Trav. Lab. Parasit. Fac. Méd.* Paris.

—— (1923a). Sur quelques infusoires nouveaux ou peu connus parasites des mammifères. *Ann. Parasit. Hum. & Comp.*, I, 209.

BULL, C. G. (1917). The pathologic effects of streptococci from cases of poliomyelitis and other sources. *Journ. Exp. Med.*, XXV, 557.

BUNDLE, A. (1895). Ciliate Infusorien im Cœcum des Pferdes. *Zeitschr. Wiss. Zool.*, LX, 284.

BUNTING, M. (1922). A preliminary note on *Tetramitus*, a stage in the life-cycle of a coprozoic amoeba. *Proc. Nat. Acad. Sci.*, VIII, 294.

BURNETT, W. I. (1851, 1852). The organic relations of some of the Infusoria, including investigations concerning the structure and nature of the genus *Bodo* (Ehr.). *Proc. Boston Soc. Nat. Hist.*, IV (1851–54), 1851, 124 (Extract); *Boston Journ. Nat. Hist.*, VI (1850–57), 1852, 319.

BUXTON, P. A. (1920). Carriage of coliform bacilli by the Oriental hornet, *Vespa orientalis* (Fabr.). *Journ. Hyg.*, XIX, 68.

—— (1920a). The importance of the house fly as a carrier of *E. histolytica*. *Brit. Med. Journ.*, I, 142.

CALKINS, G. N. (1900). *Lymphosporidium truttæ* nov. gen., nov. sp., the cause of a recent epidemic among brook trout, *Salvelinus fontinalis*. *Zool. Anz.*, XXIII, 513.

—— (1904). Studies on the life-history of Protozoa. IV. Death of the A series. Conclusions. *Journ. Exp. Zool.*, I, 423.

—— (1913). Genera and species of amoeba. *Trans. XV Internat. Congress Hyg. & Demogr.*, Washington, 1912, II, 287.

—— (1915). *Didinium nasutum.* I. The life-history. *Journ. Exp. Zool.*, XIX, 225.

—— (1919). *Uroleptus mobilis* Engelm. I. History of the nuclei during division and conjugation. *Ibid.*, XXVII, 273.

—— (1919a). *Uroleptus mobilis* Engelm. II. Renewal of vitality through conjugation. *Ibid.*, XXIX, 121.

CALKINS, G. N., & CULL, S. W. (1907). The conjugation of *Paramœcium aurelia* (*caudatum*). *Arch. Protist.*, X, 375.

CANALIS, P. (1889). *Sopra il allo evolutivo delle forme semilunari de Laveran e sulle febbri malariche irregolari e perniciose che da esse dipendono.* Rome.

CANNATA, S. (1913). Reperto del parassita di Leishman nel sangue periferico. *Pathologica*, V, 351.

—— (1913a). Inclusioni leucocitarie nella Leishmaniosi infantile. *Ibid.*, V, 420.

—— (1914). Ulteriore ricerche sulla presenza del parassita di Leishman nel sangue periferico di bambini affetti da leishmaniosi. *Pediatria*, XXII, 27.

CANNATA, S., & CARONIA, G. (1914). Cultura dei parassiti di Leishman dal sangue periferico nella leishmaniosi infantile. *Pathologica*, VI, 396.

CARDAMATIS, J. P. (1911). L'*Hœmamœba ziemanni* d'après les observations faites. *Centralbl. Bakt.*, I. Abt., LX, 241.

—— (1912). Leishmaniose du chien en Grèce. *Bull. Soc. Path. Exot.*, V, 88.

—— (1919). Le paludisme en Macédoine. *Malariologia*, XII, 65.

CARDAMATIS, J. P., & MELISSIDIS, A. (1911). Deux cas de bouton d'Orient dont le premier très rare; antagonisme probable entre le bouton d'Orient et le kala-azar. *Bull. Soc. Path. Exot.*, IV, 454.

—— (1911a). Du rôle probable de la mouche domestique dans la transmission des *Leishmania*. *Ibid.*, IV, 459.

CARINI, A. (1909). Reproduction expérimentale de la toxoplasmose du lapin. *Ibid.*, II, 465, 524.

—— (1910). Sobre a " *Hæmogregarina muris*." *Rev. Soc. Scient. S. Paulo*, V.

—— (1910a). Formas de eschizogonia do *Trypanosoma lewisi*. *Soc. Med. e Cir. S. Paulo* (August 16).

—— (1911). Leishmaniose de la muqueuse rhino-bucco-pharyngée. *Bull. Soc. Path. Exot.*, IV. 289.

—— (1911a). Infection spontanée du pigeon et du chien due au *Toxoplasma cuniculi*. *Ibid.*, IV, 518.

—— (1915). Ueber die Hundekrankheit Nambi-uvu und ihren Parasiten, *Rangelia vitalii. Centralbl. Bakt.*, I. Abt., LXXVII, 265.

CARINI, A., & MACIEL, J. (1913). Toxoplasmose naturelle du chien. *Bull. Soc. Path. Exot.*, VI, 681.

—— —— (1914). Sobre o " *Pneumocystis carinii*." *Ann. Paulist. Med. & Cir.*, II, 170.

—— —— (1914a). Sobre a molestias dos câes, chamada Nambi-Uvû e o seu parasita (*Rangelia vitalii*). *Ibid.*, III, 65.

—— —— (1915). Ueber *Pneumocystis carinii*. *Centralbl. Bakt.*, I. Abt., LXXVII, 46.

CARINI, A., & MIGLIANO, L. (1916). Sur un toxoplasme du cobaye (*Toxoplasma caviæ* n. sp.). *Bull. Soc. Path. Exot.*, IX. 435; *Ann. Paulist. Med. & Cir.*, VI, 113.

CARINI & PARANHOS, U. (1909). Identification de l'" Ulcera de Bauru " avec le bouton d'Orient. *Bull. Soc. Path. Exot.*. II, 255.

CARINI, A., & RUDOLPH, M. (1912). Sur quelques hématozoaires de lézards au Brésil. *Ibid.*, V, 592.

CARONIA, G. (1916). L'impiego di nuovi preparati di antimonio per via intramuscolare nella cura della leishmaniosi infantile. *Pediatria*, XXIV, i, 65.

CARONIA [G.] & DI GIORGIA, G. (1914). Sulla leishmaniosi spontanea nei cani di Palermo. *Pathologica*, VI, 208.

CARPANO, M. (1912). La febbra della costa nella colonia Eritrea. Note biologiche e morphologiche sulla " *Theileria parva*." *Clin. Vet.*, Milan, XXXV, 821.

—— (1913). Sul ciclo evolutivo dell' *Hæmoproteus danlewskyi*. Note morphologiche e biologiche del parassita riscontrato in alcune specie di uccelli della colonia Eritrea. [*Collez. pubb. sci. sull' Eritrea*. I. Studi di Medicina Tropicale. Bergamo. 1914, 129.]

—— (1913a). Su di un Piroplasma del tipo *parvum* (genre *Theileria*) riscontrato nella gazzella in Eritrea. *Clin. Vet.*, Milan, XXXVI, 254.

—— (1913b). Piroplasmosi equina, tipi parassitari. *Ibid.*, XXXVI, 845.

—— (1915). Piroplasmosi tipo " *parvum* " nei bovini del basso bacino del Mediterraneo. (Febbre della costa mediterranea.) *Ibid.*, XXXVIII. 497. 553.

CARSWELL, R. (1838). *Pathological anatomy. Illustrations of the elementary forms of diseases.* London.

CARTAYA, J. T. (1910). A new filaria and other parasites in the blood of the bat. *Sanid. y Benefic.*, Habana, III, 507.

CARTER, R. M. (1909). Oriental sore of Northern India, a protozoal infection. *Brit. Med. Journ.*, II, 647.

—— (1911). Non-ulcerating oriental sore: The cultural characteristics of the parasite as compared with a new similar parasite in *Erthesina fullo* (Thumb). a pentatomid bug. *Ann. Trop. Med. & Parasit.*, V, 15.

CASAGRANDI, O., & BARBAGALLO, P. (1895). Ricerche biologiche e cliniche sull' *Amœba coli* (Lösch). *Bull. Accad. Gicenia Sci. Nat.*, Catania, XXXIX (N.S.), 4; XLI (N.S.), 7.

—— —— (1897). *Entamœba hominis* s. *Amœba coli* (Lösch). Studio biologico e clinico. *Annali d'Igiene*, VII, 103.

—— —— (1907). Sulla trasmissibilità dell' infezione alteridica da animali recettivi a refrattari. *Ibid.*, XVII, 571.

CASTELLANI, A. (1903). Trypanosoma in sleeping sickness. *Brit. Med. Journ.*, I, 1218.

—— (1905). Observations on some Protozoa found in human fæces. *Centralbl. Bakt.*, I. Abt., XXXVIII, 66.

—— (1908). Note on a liver abscess of amœbic origin in a monkey. *Parasitology*, I, 101.

—— (1912). Kala-azar in dogs in Colombo. Editorial notes in *Malaria e Mal. Paesi Caldi*, III, 1912, 91; [and Castellani & Chalmers's *Manual of Tropical Diseases*, 2nd edition, 1913, 363; 3rd edition, 1919, 375.]

CASTELLANI, A. (1913). Indian oro-pharyngeal leishmaniasis. *Journ. Trop. Med. & Hyg.*, XVI, 49.
—— (1914). Note on an intestinal protozoal parasite producing dysenteric symptoms in man. *Ibid.*, XVII, 65.
—— (1914a). A further case of entoplasmosis. *Ibid.*, XVII, 83.
—— (1914b). Note on certain Protozoa-like bodies in a case of protracted fever with spleno-megaly. *Ibid.*, XVII, 113.
—— (1917). Tropical diseases in the Balkans. *Proc. Roy. Med. Soc.*, X (Sect. Med.), 31.
—— (1924). [Remarks on *Plasmodium canis*.] *Proc. Internat. Conf. Health Problems in Trop. Amer.*, Boston, Mass., 73.
CASTELLANI, A., & CHALMERS, A. J. (1910). *Manual of Tropical Medicine*. 1st Ed. Lond.
—— —— (1913). *Ibid.* 2nd edition. London.
—— —— (1919). *Ibid.* 3rd edition. London.
CASTELLANI, A., & WILLEY, A. (1904). Observations on the Hæmatozoa of vertebrates in Ceylon. *Spolia Zeylanica*, II, 78.
CASTEX, M. R., & GREENWAY, D. (1923). Coccidiasis humana por *Isospora hominis*. *Prensa Med. Argentina* (August 20).
CATANEI, A. (1923). Essai critique sur la morphologie des parasites du paludisme. *Arch. Inst. Pasteur Algérie*, I, 10.
CATOUILLARD, G. (1909). Sur un trypanosome du gecko commun de Tunisie (*Platydactylus muralis*). *C. R. Soc. Biol.*, LXVII, 804.
CAUCHEMEZ, L. (1921). Fréquence des amibes iodophiles chez le porc en France. *Bull. Soc. Path. Exot.*, XIV, 321.
—— (1921a). Fréquence de la coccidie du porc (*Eimeria brumpti*, n. sp.) en France. *Ibid.*, XIV, 645.
—— (1922). Au sujet du nom spécifique à donner à la coccidie du porc. *Ibid.*, XV, 160.
—— (1922a). Présence d'amibes non iodophiles chez des porcs français. *Ibid.*, XV, 390.
CAULLERY, M. (1906). Sur un amœbien parasite des embryons de *Peltogaster curvatus* Kossm. *C. R. Soc. Biol.*, LXI, 266.
CAULLERY, M., & MESNIL, F. (1899). Sur quelques parasites internes des annélides. *Trav. Stat. Zool.*, Wimereux, VII, 80.
—— —— (1905). Recherches sur les haplosporidies. *Arch. Zool. Exp.*, IV, 101.
—— —— (1905a). Recherches sur les actinomyxidés. *Arch. Protist.*, VI, 272.
CAVOLINI, F. (1787). *Memoria sulla generazione dei pisci e dei granchi*. Naples. (German transl. by Zimmermann, Berlin, 1792.)
CAZALBOU (1903). Note sur un trypanosome du dromadaire au Soudan français. *Bull. Acad. Méd.*, XLIX, 807.
CAZALBOU, L. (1906). Expérience d'infection de trypanosomiase par des *Glossina palpalis* infectées naturellement. *C. R. Acad. Sci.*, CXLIII, 435.
—— (1913). Observation d'un nouveau trypanosome chez le lapin. *Rec. Méd. Vét.*, XC, 155.
CELLI, A. (1900). *La malaria secondo le nueve ricerche*. 2nd edition. Rome. (English edition, 1901, London.)
CELLI, A., & FIOCCA, R. (1894). Beiträge zur Amöbenforschung, I & II. *Centralbl. Bakt.*, I. Abt., XV, 470; XVI, 329.
—— —— (1895). Ueber die Aetiologie der Dysenterie. *Ibid.*, I. Abt., XVII, 309.
CELLI, A., & MARCHIAFAVA, E. (1890). Sulle febbri malariche predominanti nell' estate e nell' autunno in Roma. *Arch. Sci. Med.*, XIV, 177.
CELLI, A., & SAN FELICE, F. (1891). Sul parassita del globo rosso nel' uomo e negli animali. *Ann. Agric.*, Rome, No. 138, 41; *Fortschr. Med.*, IX, 499, 541, 581.
CÉPÈDE, C. (1907). Sur un nouvel infusoire astome, parasite des testicules des étoiles du mer. Considérations générales sur les *Astomata*. *C. R. Assoc. Franç. Avanc. Sci.*, XXXVI, 258.
—— (1910). Recherches sur les infusoires astomes. *Arch. Zool. Exp.*, III, 341.
—— (1924). *Mrazekia piscicola* n. sp. Microsporidie parasite du merlan *Gadus merlangus* Linné. *Bull. Soc. Zool.*, Paris, XLIX, 109.
CERTES, A. (1889). Note sur les micro-organismes de la panse des ruminants. *Ibid.*, XIV, 70.
CÉSARI (1925). La leishmaniose canine en France. *Rec. Méd. Vét.*, CI, 177.
CHAGAS, C. (1909). Ueber eine neue Trypanosomiasis des Menschen. Studien über Morphologie und Entwicklungszyklus des *Schizotrypanum cruzi*, n. gen., n. sp., Erreger einer nuen Krankheit des Menschen. *Mem. Inst. O. Cruz*, I, 159.
—— (1910). Estudos de citolojia em nova especie de Coccidio, *Adelea hartmanni*, do intestino do *Dysdercus ruficollis* L. (and in German). *Ibid.*, II, 168.
—— (1911). Sobre as variações ciclicas do cariozoma em duas especies de ciliados parazitos. Contribuição para o estudo do nucleo nos infuzorios. *Ibid.*, III, 136.
—— (1912). Sobre um trypanosoma do tatú, *Tatusia novemcincta*, transmittido pela *Triatoma geniculata* Latr. (1811). Possibilidade de ser o tatú um depositario do *Trypanosoma cruzi* no mundo exterior. *Brazil-Medico*, XXVI, 305.

CHAGAS, C. (1913). Rivisão do cyclo evolutivo do *Trypanosoma cruzi*. *Ibid.*, XXVII, 225.

—— (1918). [Host of *Trypanosoma cruzi*.] *Rev. Med. Cirurg.*, Brazil, XXVI, 220.

—— (1924). Infection naturelle des singes du Para (*Crysothrix sciureus* L.) par *Trypanosoma cruzi*. *C. R. Soc. Biol.*, XC, 873.

CHAGAS, C., & VIANNA, G. (1911). Le cycle de *Schizotrypanum cruzi* chez l'homme et les animaux de laboratoire. *Bull. Soc. Path. Exot.*, IV, 467.

CHALACHNIKOV (1888). *Recherches sur les parasites du sang des animaux à sang chaud et à sang froid.* Kharkov.

CHALMERS, A. J. (1918). The classification of trypanosomes. *Journ. Trop. Med. & Hyg.*, XXI, 221.

CHALMERS, A. J., & ARCHIBALD, R. G. (1920). The " tenue " phase of *Plasmodium vivax* (Grassi and Feletti, 1890). *Ibid.*, XXIII, 33.

CHALMERS, A. J., & KAMAR, A. (1920). *Toxoplasma pyrogenes* Castellani, 1913. *Ibid.*, XXIII, 45.

CHALMERS, A. J., & PEKKOLA, W. (1916). A new human intestinal flagellate in the Anglo-Egyptian Sudan. *Ibid.*, XIX, 142.

—— —— (1917). *Chilomastix mesnili* (Wenyon, 1910). *Ann. Trop. Med. & Parasit.*, XI, 213.

—— —— (1917a). *Enteromonas hominis* da Fonseca, 1915. *Trans. Soc. Trop. Med. & Hyg.*, XI, 93.

—— —— (1918). *Enteromonas hominis* and *Protetramitus testudinis*. *Journ. Trop. Med. & Hyg.*, XXI, 129.

—— —— (1919). *Dicercomonas soudanensis*. *Ibid.*, XXII. 29.

—— —— (1919a). *Diplocercomonas soudanensis*. *Ibid.*, XXII, 190.

CHATTERJEE, G. C. (1915). On a five-flagellate *Trichomonas* (n. sp.) parasitic in man. *Indian Med. Gaz.*, L, 5.

—— (1917). Note on flagellate dysentery. *Indian Journ. Med. Res.*, IV, 393.

—— (1917a). A *Trichomastix* (n. sp.) parasitic in the human intestine. *Ibid.*, V, 217.

—— (1919). On an *Enteromonas* n. sp. found in the human intestinal contents. *Ibid.*, VI. 380.

—— (1920). An atypical amœba causing dysenteric lesions. *Philippine Journ. Sci.*, XVII. 385.

—— (1923). On a *Tetrachilomastix* n. sp. parasitic in human intestine. *Indian Journ. Med. Res.*, XI. 177; *Arch. Protist.*, XLVI, 373.

CHATTON, E. (1909). Une amibe, *Amœba mucicola* n. sp., parasite des branchies des labres, associée à une trichodine. (Note préliminaire.) *C. R. Soc. Biol.*, LXVII, 690.

—— (1909a). Sur un trypanosomide nouveau, *Leptomonas agilis*, d'une réduve indigène (*Harpactor iracundus* Scop.). *Ibid.*, LXVI, 981.

—— (1910). Le kyste de Gilruth dans la muqueuse stomacale des ovidés. *Arch. Zool. Exp.*, V. (N. & R.), cxiv.

—— (1910a). A propos de " *Rhynchomonas luciliæ* " Patton, flagellé parasite de la lucilie sérénissime. *Bull. Soc. Path. Exot.*, III, 363.

—— (1910b). Protozoaires parasites des branchies des labres. *Amœba mucicola* Chatton, *Trichodina labrorum* n. sp. *Arch. Zool. Exp.*, V, 239.

—— (1910c). Essai sur la structure du noyau et la mitose chez les amœbiens. Faits et théories. *Ibid.*, V, 267.

—— (1911). Sur la systématique des trypanosomides des insectes. *C. R. Soc. Biol.*, LXXI, 578.

—— (1911a). Microsporidies considérées comme causes d'erreurs dans l'étude du cycle évolutif des trypanosomides chez les insectes. *Ibid.*, IV, 662.

—— (1912). *Leptomonas* de deux *Borborinæ* (Muscides). Evolution de *L. legerorum* n. sp. *Ibid.*, LXXIII, 286.

—— (1912a). *Leptomonas roubaudi* n. sp., parasite des tubes de Malpighi de *Drosophila confusa* Staeger. *Ibid.*, LXXIII, 289.

—— (1912b). Sur un *Coccidium* de deux *Cerastes* et sur une adéléidée trouvée dans l'intestin de *Scincus officinalis*. *Bull. Soc. Zool.*, Paris, XXXVII, 8.

—— (1912c). [Review of paper by Gilruth and Bull, 1912.] *Bull. Inst. Pasteur*, X. 792.

—— (1913). Position systématique et signification phylogénique des trypanosomes malpighiens des muscides. Le genre *Rhynchoidomonas* Patton. *C. R. Soc. Biol.*, LXXIV, 551.

—— (1913a). L'ordre, la succession et l'importance relative des stades, dans l'évolution des trypanosomides, chez les insectes. *Ibid.*, LXXIV, 1145.

—— (1917). Réalisation expérimentale chez le cobaye de l'amibiase intestinale à *Entamœba dysenteriæ*. *Bull. Soc. Path. Exot.*, X, 794.

—— (1917a). L'éclosion des kystes et les premiers stades de l'évolution de l'amibe dysentérique humaine chez le chat. *Ibid.*, X, 834.

—— (1917b). Les " *Blastocystis* " stades du cycle évolutif de flagellés intestinaux. *C. R. Soc. Biol.*, LXXX, 555.

—— (1918). Le laboratoire militaire de bactériologie du Sud Tunisien (à Gabès). *Arch. Inst. Pasteur Tunis*, X, 199.

—— (1918a). Présence d'un flagellé intestinal du genre *Trichomastix* dans le sang et les organes du gecko: *Tarentola mauritanica*. Cultures sur gélose au sang N.N.N. *C. R. Soc. Biol.*, LXXXI, 343.

CHATTON, E. (1918b). Culture pure indéfinie d'un flagellé intestinal du genre *Trichomastix* sur organes en autolyse aseptique. *Ibid.*, LXXXI, 346.

—— (1918c). Les caractères de l'amibiase intestinale du cobaye à *Entamœba dysenteriœ :* localisation cæcale, absence de dysenterie, importantes réactions hyperplasiques. *Bul. Soc. Path. Exot.*, XI, 23.

—— (1918d). L'amibiase intestinale expérimentale du cobaye à *Entamœba dysenteriœ*. *Arch. Inst. Pasteur Tunis*, X, 138.

—— (1919). Sur la culture pure d'un leptomonas de la puce du chien et sur un caractère de ces formes culturales qui les distinguent de celles du kala-azar de souches humaine et canine. *Bull. Soc. Path. Exot.*, XII, 313.

—— (1920). Culture indéfinie d'un *Trichomonas* intestinal du cobaye, essais de purification, action morphogène des milieux sur la membrane ondulante. *C. R. Soc. Biol.*, LXXXIII, 69.

—— (1924). Sur un *Leptomonas* d'un nematode marin et la question de l'origine des trypanosomides. *Ibid.*, XC, 780.

CHATTON, E., & ALILAIRE, E. (1908). Coexistence d'un *Leptomonas* (*Herpetomonas*) et d'un *Trypanosoma* chez un muscide non vulnérant, *Drosophila confusa* Staeger. *Ibid.*, LXIV, 1004.

CHATTON, E., & AUBERTOT, M. (1924). Sur les phases et les voies d'extension des infections à *Leptomonas* intestinaux des drosophiles. La non spécificité parasitaire du *Leptomonas drosophilæ*. *Ibid.*, XCI. 283.

CHATTON, E., & AVEL, M. (1923). Sur la sarcosporidie du gecko et ses cytophanères. La paroi et le réticulum sarcosporidiens appartiennent en propre au parasite. *Ibid.*, LXXXIX, 181.

CHATTON, E., & BLANC, G. (1914). Existence de corps leishmaniformes dans les hématoblastes d'un gecko barbaresque, *Tarentola mauritanica* L. Gunth. *Ibid.*, LXXVII, 430.

—— —— (1914a). Sur un hématozoaire nouveau *Pirhemocyton tarentolæ*, du gecko (*Tarentola mauritanica*) et sur les altérations globulaires qu'il détermine. *Ibid.*, LXXVII, 496.

—— —— (1916). Précisions sur la morphologie de l'hématozoaire endoglobulaire de la tarente: *Pirhemocyton tarentolæ* Chatton et Blanc. *Ibid.*, LXXIX, 39.

—— —— (1917). Notes et réflexions sur le toxoplasme et la toxoplasmose du gondi (*Toxoplasma gundii* Ch. Nicolle et Manceaux, 1909). *Arch. Inst. Pasteur Tunis*, X, 1.

—— —— (1918). Large éclectisme parasitaire de la punaise des lits. Son entretien aux dépens des reptiles. *Bull. Soc. Path. Exot.*, XI, 382.

—— —— (1918a). Culture du trypanosome du gecko chez la punaise des lits. *Ibid.*, XI, 387.

—— —— (1918b). Le *Leptomonas* de la tarente dans une région indemne de bouton d'orient. Observations et expériences. *Ibid.*, XI, 595.

CHATTON, E., & BRODSKY, A. (1909). Le parasitisme d'une chytridinée du genre *Sphærita* Dangeard chez *Amœba limax* Dujard. Étude comparative. *Arch. Protist.*, XVII, 1.

CHATTON, E., & COURRIER, R. (1921). Un *Schizotrypanum* chez les chauve-souris (*Vesperugo pipistrellus*) en Basse-Alsace. Schizotrypanose et goitre endémique. *C. R. Soc. Biol.*, LXXXIV, 943.

CHATTON, E., & DELANOË, P. (1912). Observations sur l'évolution et la propagation de *Crithidia melophagi* Flu. *Ibid.*, LXXII, 942.

—— —— (1912a). *Leptomonas pattoni* (Swingle) et *Tr. lewisi* (Kent) chez l'adulte et la larve de *Ceratophyllus fasciatus*. *Ibid.*, LXXIII, 291.

CHATTON, E., & KREMPF, A. (1911). Sur le cycle évolutif et la position systématique des protistes du genre " *Octosporea* " Flu parasites des muscides. *Bull. Soc. Zool.*, Paris, XXXVI, 172.

CHATTON, E., & LALUNG-BONNAIRE (1912). Amibe limax (*Vahlkampfia* n. gen.) dans l'intestin humain. Son importance pour l'interprétation des amibes de culture. *Bull. Soc. Path. Exot.*, V, 135.

CHATTON, E., & LEGER, A. (1911). Eutrypanosomes, leptomonas et leptotrypanosomes chez *Drosophila confusa* Staeger (muscide). *C. R. Soc. Biol.*, LXX, 34.

—— —— (1911a). Sur quelques leptomonas de muscides et leurs leptotrypanosomes. *Ibid.*, LXX, 120.

—— —— (1911b). Sur l'autonomie spécifique du *Trypanosoma drosophilæ* Chatton et Alilaire, et sur les eutrypanosomes des muscides non sanguivores. *Ibid.*, LXXI, 573.

—— —— (1911c). Documents en faveur de la pluralité des espèces chez les leptomonas des drosophiles. Remarques sur leur morphologie. *Ibid.*, LXXI, 663.

—— —— (1912). Diversité des formes de reproduction chez les trypanosomides des insectes. *Ibid.*, LXXII, 20.

CHATTON, E., & LEGER, A. & M. (1912). Trypanosomides et membrane péritrophique chez les drosophiles. Culture et évolution. *Ibid.*, LXXII, 453.

CHATTON, E., & LEGER, M. (1911). Sur l'axostyle ou axoplaste des trypanosomides des insectes. *Ibid.*, LXXI, 575.

—— —— (1912). Sur un mode particulier d'agglutination et de cytolyse simulant un enkystement chez les leptomonas des drosophiles. *Ibid.*, LXXII, 171.

—— —— (1912a). Du déterminisme des infections endotrophiques ou péritrophiques des drosophiles par leur trypanosomides. Infections larvaires et imaginales. *Ibid.*, LXXII, 550.

CHATTON, E. & LEGER, M. (1913). L'autonomie des trypanosomes propres aux muscides démontrée par les élevages purs indéfinis. *Ibid.*, LXXIV, 549.

CHATTON, E., & PÉRARD, C. (1919). *Nicollella ctenodactyli* n. g., n. sp., et *Collinella gondii* n. g., n. sp., ciliés parasites intestinaux du gondi, *Ctenodactylus gundi* Pellas (rongeur). La famille des Nicollellidæ nov. fam. Note préliminaire. *Bull. Soc. Zool.*, Paris, XLIV, 10.

—— —— (1921). Les Nicollellidæ, infusoires intestinaux des gondis et des damans, et le "cycle évolutif des ciliés." *Bull. Biol. France & Belgique*, LV, 87.

CHATTON, E., & ROUBAUD, E. (1913). Sporogonie d'une hémogrégarine chez une tsétsé (*Glossina palpalis* R. Desv.). *Bull. Soc. Path. Exot.*, VI, 226.

CHAUSSAT, J. B. (1850). Des hématozoaires. (Thèse pour le doctorat en médecine.) Paris.

CHIANG, S. F. (1925). The rat as a possible carrier of the dysentery amœba. *Proc. Nat. Acad. Sci.*, Washington, II, 239.

CHIERICI, G. (1908). Note di parassitologia. La coccidiosi epatica nel gatto. *Nuovo Ercolani*, Pisa, XIII, 54.

CHIMISSO, L. (1922). Su un plasmodio della malaria di scimmia (*Macacus rheus*). *Hæmatologica*, III.

CHRISTELLER, E. (1922). Über die Balantidienruhr bei den Schimpansen des Berliner Zoologischen Gartens. *Virchow's Archiv*, CCXXXVIII, 396.

CHRISTOPHERS, S. R. (1904). A preliminary report on a parasite found in persons suffering from enlargement of the spleen in India. *Sci. Mem. Off. Med. & San. Dep. Gov. India*. N.S., VIII.

—— (1905). *Hæmogregarina gerbilli*. *Ibid.*, N.S., XVIII.

—— (1906). *Leucocytozoon canis*. *Ibid.*, N.S., XXVI.

—— (1907). Preliminary note on the development of the *Piroplasma canis* in the tick. *Brit. Med. Journ.*, I, 76.

—— (1907a). The sexual cycle of *Leucocytozoon canis* in the tick. *Sci. Mem. Off. Med. & San. Dep. Gov. India*, N.S., XXVIII.

—— (1907b). *Piroplasma canis* and its life-cycle in the tick. *Ibid.*, N.S., XXIX.

—— (1912). The development of *Leucocytozoon canis* in the tick, with a reference to the development of piroplasma. *Parasitology*, V. 37.

—— (1922). Kala-azar inquiry of the Indian Research Fund Association. *Indian Journ. Med. Res.*, IX, v.

CHRISTOPHERS, S. R., SHORTT, H. E., & BARRAUD, P. J. (1925). The development of the parasite of Indian kala-azar in the sand fly, *Phlebotomus argentipes* Annandale and Brunetti. *Ibid.*, XII, 605.

—— —— —— (1925a). Further observations on the feeding of sand flies, *Phlebotomus argentipes*, on cases of Indian kala-azar. *Ibid.*, XIII, 159.

—— —— —— (1925b). Culicoides and kala-azar in Assam. *Ibid.*, XIII, 175.

CHRISTOPHERSON, J. B. (1914). On a case of naso-oral leishmaniasis (corresponding to the description of espundia), and a case of oriental sore, both originating in the Anglo-Egyptian Sudan. *Ann. Trop. Med. & Parasit.*, VIII, 485.

CIENKOWSKI, L. (1875). Ueber einige Rhizopoden und verwandte Organismen. *Arch. Mikr. Anat.*, XII, 15.

CIUCA, A. (1913). A propos de l'immunité active du chien vis-à-vis de la piroplasmose canine (babesiose canine). *Bull. Soc. Path. Exot.*, VI, 499.

CLAPARÈDE, E., & LACHMANN, J. (1858). *Etudes sur les infusoires et les rhizopodes.* Geneva.

CLAPIER, P. (1921). Constatation de *Trypanosoma gambiense* en grande abondance dans le liquide d'hydrocèle. *Bull. Soc. Path. Exot.*, XIV, 378.

CLARK, H. C. (1918). Piroplasmosis of cattle in Panama. *Journ. Infect. Dis.*, XXII, 159.

CLARK, H. C., & ZETEK, J. (1925). Tick-biting experiments in bovine and cervine piroplasmosis. *Amer. Journ. Trop. Med.*, V, 17.

CLARKE, J. (1895). A study of coccidia met with in mice. *Quart. Journ. Micro. Sci.*, XXXVII, 277.

CLELAND, J. B. (1906). The hæmogregarine of mammals (*H. balfouri*) and some notes on rats. *Journ. Trop. Med. & Hyg.*, IX, 296.

CLEVELAND, L. R. (1923). Symbiosis between termites and their intestinal Protozoa. *Proc. Acad. Nat. Sci. Philadelphia*, IX, 424.

COHN, L. (1896). Über die Myxosporidien von *Esox lucius* und *Perca fluviatilis*. *Zool. Jahrb.*, Abt. Anat., IX, 227.

COHNHEIM, P. (1903). Ueber Infusorien im Magen und im Darmkanal des Menschen und ihre klinische Bedeutung. *Deutsch. Med. Woch.*, XXIX, 206, 230, 245.

—— (1909). Infusorien bei gut- und bösartigen Magenleiden nebst Bemerkungen über die sogenannte Infusorienenteritis. *Ibid.*, XXXV. 92.

COLE, L. J., & HADLEY, P. B. (1910). Blackhead in turkeys: A study in avian coccidiosis. *Rhode Isl. Agric. Exp. Stat.*, Bull. No. 124, 67.

COLES, A. C. (1914). Blood parasites found in mammals, birds, and fishes in England. *Parasitology*, VII, 17.

COLLIER, J., & BOECK, W. C. (1926). The morphology and cultivation of *Embadomonas cuniculi* n. sp. *Journ. Parasit.*, XII, 131.

COLLIN, B. (1909). La conjugaison d'*Anoplophrya branchiarum* (Stein) (*A. circulans* Balbiani). *Arch. Zool. Exp.*, I, 345.

—— (1913). Sur un ensemble de protistes parasites des batraciens. (Note préliminaire.) *Ibid.*, LI (N. & R.), lix.

COLLIN. B. (1914). Notes protistologiques. *Ibid.*, LIV, (N. & R.), 85.

COMES, S. (1912). Riproduzione e morfologia di *Dinenympha gracilis* Leidy flagellato ospite dell'intestino dei termitidi. *Arch. Protist.*, XXV, 275.

CONDORELLI, M., & FIORE, C. DE (1892). Un caso di psorospermosi intestinale nel *Coccothraustes vulgaris*. *Boll. Soc. Rom. Studi Zoolog.*, I, 68.

CONNAL, A. (1917). *Report of the Medical Research Institute, Lagos, for the year 1916.*

—— (1922). Observations on the pathogenicity of *Isospora hominis*, Rivolta, emend. Dobell, based on a second case of human coccidiosis in Nigeria, with remarks on the significance of Charcot-Leyden crystals in the fæces. *Trans. Roy. Soc. Trop. Med. & Hyg.*, XVI, 223.

CONNAL, A., & YOUNG, W. A. (1922). A case of human coccidiosis in Nigeria. *Ibid.*, XVI, 90.

CONNOR, F. P., & SHORTT. H. E. (1918). *Leishmania tropica* infections in Mesopotamia, with a note on diagnostic and cultural methods. *Indian Journ. Med. Res.*, VI, 162.

CORNWALL, J. W., & LA FRENAIS, H. M. (1916). A contribution to the study of kala-azar—I. *Ibid.*, III. 698.

—— —— (1922). A contribution to the study of kala-azar—V. *Ibid.*, IX, 533.

COUNCILMAN, W. T., & LAFLEUR, H. A. (1891). Amœbic dysentery. *Johns Hopkins Hosp. Rept.*. II, 395.

COVENTRY, F. A. (1925). The reaction product which inhibits the reproduction of the trypanosomes in infections with *Trypanosoma lewisi*, with special reference to its changes in titre throughout the course of the infection. *Amer. Journ. Hyg.*, V, 127.

COWDRY, E. V. (1923). The distribution of *Rickettsia* in the tissues of insects and arachnids. *Journ. Exp. Med.*, XXXVII, 431.

—— (1925). Studies on the etiology of heartwater—I and II. *Ibid.*, XLII, 231 and 253.

COWDRY, E. V., & NICHOLSON, F. M. (1924). Meningo-encephalitic lesions and Protozoan-like parasites. *Journ. Amer. Med. Assoc.*, LXXXII, 545.

CRAGG, F. W. (1917). Observations on dysentery cases from Mesopotamia. *Indian Journ. Med. Res.*, V. 301.

—— (1919). A contribution to our knowledge of *Entamœba coli*. *Ibid.*, VI, 462.

CRAIG, C. F. (1905). Observations upon amœbas infecting the human intestine, with a description of two species, *Entamœba coli* and *Entamœba dysenteriæ*. *American Medicine*, Philadelphia, IX, 854, 897, 936.

—— (1906). A new intestinal parasite of man—*Paramœba hominis*. *Amer. Journ. Med. Sci.*, CXXXII, 214.

—— (1912). *The parasitic amœbæ of man*. Philadelphia.

—— (1916). Observations on the endamœbæ of the mouth. I. *Endamœba gingivalis (buccalis)*. *Journ. Infect. Dis.*, XVIII, 220.

CRAWLEY, H. (1905). *Cœlosporidium blattellæ*. a new sporozoan parasite of *Blattella germanica*. *Proc. Acad. Nat. Sci. Philadelphia*. LVII, 158.

—— (1909). Studies on blood and blood parasites. I. Observations on mammalian blood with dark-field illumination. II. The priority of *Cryptobia* Leidy, 1846, over *Trypanoplasma* Laveran and Mesnil, 1901. III. *Trypanosoma americanum* n. sp., a trypanosome which appears in cultures made from the blood of American cattle. (Preliminary notice). *U.S. Dept. Agric., Bur. Anim. Indust.*, Bull. 199, 5.

—— (1912). *Trypanosoma americanum*, a common blood parasite of American cattle. *Ibid.*, Bull. 145.

—— (1916). The sexual evolution of *Sarcocystis muris*. *Proc. Acad. Nat. Sci. Philadelphia*, LXVIII, 2.

—— (1924). Evolution in the ciliate family Ophryoscolecidæ. *Ibid.*, LXXV, 393.

CREECH, G. T. (1922). Sarcosporidiosis of swine associated with advanced degenerative changes in the musculature. *Journ. Amer. Vet. Med. Assoc.*, LXI, 383.

CRISTINA, G. DI, & CARONIA, G. (1913). Ricerche serologiche nella leishmaniosi infantile. *Pediatria*, XXI, 801.

CRITIEN, A. (1910). Kala-azar infantile à Malte. *Arch. Inst. Pasteur Tunis*. V, 49.

—— (1911). Infantile leishmaniasis (marda tal biccia)in Malta. *Ann. Trop. Med. & Parasit.*,V,37.

CROOKSHANK, E. M. (1886). Flagellated Protozoa in the blood of diseased and apparently healthy animals. *Journ. R. Micro. Scc.*, VI, ii, 913.

CROSS, H. E. (1920). A note on the treatment of surra in camels by intravenous injections of tartar emetic. *Agric. Res. Inst. Pusa*, Bull. No. 95.

—— (1920a). The treatment of surra in camels by intravenous injections of tartar emetic. *Dept. Agric., Punjab*, Vet. Bull. No. 2.

—— (1921). The course that surra runs in camels when naturally contracted and when artificially inoculated. *Agric. Res. Inst. Pusa*, Bull. No. 98.

CROSS, H. E. (1921a). The course that surra runs in ponies, buffaloes, and other animals. *Ibid.*, Bull. No. 99.

—— (1923). A further note on surra transmission experiments with *Tabanus albimedius* and ticks. *Dept. Agric., Punjab*, Vet. Bull. No. 12; *Trans. Roy. Soc. Trop. Med. & Hyg.*, XVI, 469.

CROSS, H. E., & PATEL, P. G. (1921). A note on the transmission of surra by ticks. *Dept. Agric., Punjab*, Vet. Bull. No. 6.

CROVERI, P. (1919). Osservazione sulla biologia della " *Glossina pallidipes* " della Somalia Italiana e sulla trasmissione agli animali domestici della trypanosi detta " Ghendi." *Annali d'Igiene*, XXIX, 432.

—— (1920). *Trypanosoma theileri* varietas *somalensis* chez les bovidés somaliens, considéré par rapport à la peste bovine et aux séro-vaccinations anti-pesteuses. *Rev. Path. Comp.*, XX, 183.

CROWELL, B. C. (1923). *Trypanosoma cruzi* in the tissues of the armadillo. *Journ. Amer. Med. Assoc.*, LXXX, 910.

CUÉNOT, L. (1901). Recherches sur l'évolution et la conjugaison des grégarines. *Arch. Biol.*, XVII, 581.

—— (1902). *Legerella testiculi* n. sp., coccidie parasite du testicule de *Glomeris*. *Arch. Zool. Exp.*, X (N. & R.), xlix.

CUNHA, A. DA (1914). Contribuição para o conhecimento dos ciliados parasitos dos mammiferos brasileiros. (Thesa para livra docentia.) Rio de Janeiro.

—— (1914a). Sobre os ciliados intestinais dos mamiferos—II. *Mem. Inst. O. Cruz*, VI, 212.

—— (1915). Sobre a presença de " *Selenomonas* " no cœcum dos roedores. *Brazil-Medico*, XXIX, 33.

—— (1917). Sobre a presença do *Balantidium* no cavallo. *Ibid.*, XXXI. 337.

—— (1918). Sobre os ciliados intestinaes dos mammiferos. *Ibid.*, XXXII, 161.

—— (1919). Sobre os ciliados intestinaes dos mammiferos. *Mem. Inst. O. Cruz*, XI, 5.

—— (1922). Sobre um flagellado parasito do gato. (Nota prévia.) *Brazil-Medico*, XXXVI, 285.

CUNHA, A. DA, & FONSECA, O. DA (1917). Sobre uma nova entaméba *Entamœba serpentis* n. sp. *Ibid.*, XXXI, 279.

—— —— (1918). Sobre a *Entamœba serpentis*. *Mem. Inst. O. Cruz*, X, 95.

CUNHA, A. DA, & MUNIZ, J. (1922). Sobre os flagellados intestinaes das aves, *Octomitus avium* n. sp. *Brazil-Medico*, XXXVI, ii, 386.

—— —— (1923). Parasitismo de *Trichomonas* por Chytridaceæ do genero *Sphærita* Dangeard. *Ibid.*, XXXVII, i, 19.

—— —— (1925). Sur les flagellés parasites des oiseaux brésiliens. *C. R. Soc. Biol.*, XCIII, 869.

CUNHA, A. DA, & PACHECO, G. (1923). Recherches sur les flagellés intestinaux de l'homme. *Ibid.*, LXXXIX, 765.

CUNHA, A. DA, & TORRES, C. DE M. (1924). Sur un nouveau *Globidium, Gl. tatusi* Cunha e Torres, 1923, parasite de l'armadille. *Ibid.*, XC, 242.

CUNNINGHAM, D. D. (1871). A report on cholera. Calcutta. (From *Ann. Rept. San. Commr. with Govt. India*, App. B, 141.)

—— (1885). On the presence of peculiar parasitic organisms in the tissue of a specimen of Delhi boil. *Sci. Mem. Med. Off. Army India*, I, 21.

CUNNINGHAM, J., & PUNDIT, S. R. (1925). A new endemic focus of kala-azar in Southern India. *Indian Journ. Med. Res.*, XII, 743.

CURSON, H. H. (1926). [Leishmania in the goat.] (Verbal communication).

CURTICE, C. (1892). Parasites. *Journ. Comp. Med. & Vet. Arch.*, New York, XIII, 225.

CUTLER, D. W. (1918). A method for the cultivation of *Entamœba histolytica*. *Journ. Path. & Bact.*, XXII, 22.

—— (1919). Observations on the Protozoa parasitic in the hind-gut of *Archotermopsis wroughtoni* Desn. Part I.—*Ditrichomonas* (*Trichomonas*) *termitis* Imms. *Quart. Journ. Micro. Sci.*, LXIII, 555.

DADAY, E. (1905). *Nyctotherus piscicola* n. sp., ein neuer Fischendoparasit aus Südamerika. *Zool. Anz.*, XXIX, 233.

DAKIN, W. J. (1911). Notes on a new coccidian (*Merocystis kathæ* n. gen. et sp.) occurring in the renal organ of the whelk. *Arch. Protist.*, XXIII, 145.

DALE, H. H. (1924). Progress and prospects in chemotherapy. *Brit. Med. Journ.*, II, 219.

DALE, H. H., & DOBELL, C. (1917). Experiments on the therapeutics of amœbic dysentery. *Journ. Pharmac. & Exp. Therap.*, X, 399.

DALE, T. H. (1903). Piroplasmosis of the donkey. *Journ. Comp. Path. & Therap.*, XVI, 312.

DALLINGER, W. H., & DRYSDALE, J. (1873). Further researches into the life-history of the monads. *Trans. R. Micro. Soc.*, X, 245.

DANGEARD, P. A. (1886). Recherches sur les organismes inférieurs. *Ann. Sci. Nat.* (Bot.), IV, 241.

—— (1896). Mémoire sur les parasites du noyau et du protoplasma. *Botaniste.*, IV (1894-95), 199.

DANGEARD, P. A. (1896a). Contributions à l'étude des acrasiées. *Botaniste*, V, 1.

—— (1900). Etude de la karyokinèse chez l'*Amœba hyalina* sp. nov. *Ibid.*, VII, 49.

DANIELS (1899). *Report to the Secretary of the Malaria Investigation Committee of the Royal Society, London, on the results of observations made by him in Calcutta in conjunction with Major Ross, I.M.S.* Calcutta.

DANIELS, C. W. (1899a). On transmission of *Proteosoma* to birds by the mosquito. *Proc. Roy. Soc.*, LXIV, 443; *Repts. Malar. Cttee. Roy. Soc.*, 1.

—— (1901). Development of "crescents" in the "small dark" anopheles prevalent in British Central Africa. *Repts. Malar. Cttee. Roy. Soc.*, 41.

DANILEWSKY, B. (1885). Die Hämatozoen der Kaltblüter. *Arch. Mikr. Anat.*, XXIV, 588.

—— (1885a). Zur Parasitologie des Blutes. *Biol. Centralbl.*, V, 529.

—— (1886). Zur Frage über die Identität der pathogenen Blutparasiten des Menschen und der Hæmatozoen des gesunden Tiere. *Centralbl. Med. Wiss.*, XXIV, 737, 753.

—— (1888). *Recherches sur la parasitologie comparée du sang des oiseaux.* Kharkov.

—— (1889–1891). *La parasitologie comparée du sang. I. Nouvelles recherches sur les parasites du sang des oiseaux. II. Recherches sur les hématozoaires des tortues.* Kharkov.

—— (1890). Sur les microbes de l'infection malarique aiguë et chronique chez les oiseaux et chez l'homme. *Ann. Inst. Pasteur*, IV, 753.

—— (1891). Contribution à l'étude de la microbiose malarique. *Ibid.*, V, 758.

DARLING, S. T. (1906). A protozoön general infection producing pseudotubercles in the lungs and focal necroses in the liver, spleen, and lymph nodes. *Journ. Amer. Med. Assoc.*, XLVI, 1283.

—— (1909). Sarcosporidiosis, with report of a case in man. *Arch. Intern. Med.*, III, 183.

—— (1909a). Transmission of malarial fever in the Canal Zone by anopheles mosquitoes. *Journ. Amer. Med. Assoc.*, LIII, 2051.

—— (1910). Equine trypanosomiasis in the Canal Zone. *Bull. Soc. Path. Exot.*, III, 381.

—— (1910a). Experimental sarcosporidiosis in the guinea-pig and its relation to a case of sarcosporidiosis in man. *Journ. Exp. Med.*, XII, 19.

—— (1912). Experimental infection of the mule with *Trypanosoma hippicum* by means of *Musca domestica*. *Ibid.*, XV, 365.

—— (1912a). Some blood parasites (*Hæmoproteus* and *Hæmogregarina*). *Bull. Soc. Path. Exot.*, V, 71.

—— (1913). Equine piroplasmosis in Panama. *Journ. Infect. Dis.*, XIII, 197.

—— (1914). The *Endotrypanum* of Hoffman's sloth. *Journ. Med. Res.*, XXXI, 195.

—— (1915). Entamœbic dysentery in the dog. *Proc. Med. Assoc. Isthm. Canal Zone*, VI, i, 60.

—— (1919). Sarcosporidiosis in an East Indian. *Journ. Parasit.*, VI, 98.

—— (1920). Experimental inoculation of malaria by means of *Anopheles ludlowi*. *Journ. Exp. Med.*, XXXII, 313.

DAVAINE, C. (1854). Sur des animalcules infusoires trouvés dans les selles de malades atteints du choléra et d'autres affections. *C. R. Soc. Biol.*, I, 129.

—— (1860). *Traité des entozoaires.* Paris.

—— (1875). Monadiens. *Dict. Encycl. Sci. Méd.*, (II Sér.), IX, 115.

DAVEY, J. B. (1916). Examination of the blood of wild game shot in the free shooting area of Ngara sub-district. *Ann. Med. Rept. Health San. Condit. Nyasaland Protect.*, 1915, 64.

DAVIS, H. S. (1916). The structure and development of a myxosporidian parasite of the squeteague, *Cynoscion regalis*. *Journ. Morph.*, XXVII, 333.

—— (1923). Observations on an intestinal flagellate of trout. *Journ. Parasit.*, IX, 153.

DAVIS, N. C., & REICH, W. W. (1924). Notes on coccidial oöcysts from domestic animals in California. *Ibid.*, X, 137.

DAWSON, J. A. (1919). An experimental study of an amicronucleate *Oxytricha*. *Journ. Exp. Zool.*, XXIX, 473.

—— (1920). An experimental study of an amicronucleate *Oxytricha*. *Ibid.*, XXX, 129.

DEBAISIEUX, P. (1911). Recherches sur les coccidies. I. *Klossia helicina* A. Schneider. *La Cellule*, XXVII, 87.

—— (1912). Recherches sur les coccidies. II. *Adelea ovata* A. Schneider. III. *Coccidium lacazei* Schaud. *Ibid.*, XXVII, 257.

—— (1919). Quelques protozoaires parasites des chitons et des patelles. *C. R. Soc. Biol.*, LXXXII, 1400.

—— (1919a). Microsporidies parasites des larves de Simulium: *Thelohania varians*. *La Cellule*, XXX, 47.

DEBAISIEUX, P., & GASTALDI, L. (1919). Les microsporidies parasites des larves de *Simulium*—II. *Ibid.*, XXX, 187.

DELANOÉ, P. (1911). Sur l'existence des formes trypanosomes dans les cultures de *T. lewisi*. *C. R. Soc. Biol.*, LXX, 704.

—— (1912). Sur les rapports des kystes de Carini du poumon des rats avec le *Trypanosoma lewisi*. *C. R. Acad. Sci.*, CLV, 658.

DELANOË, P. (1920). Un cas d'infection spontanée du chien par *Trypanosoma marocanum*, Sergent, Lhéritier et Belleval, 1915. *Bull. Soc. Path. Exot.*, XIII, 23.

DELANOË, P., & MME. (1914). De la rareté de *Pneumocystis carinii* chez les cobayes de la région de Paris. Absence de kystes chez d'autres animaux (lapins, grenouille, 3 anguilles). *Ibid.*, VII, 271.

DELANOË, P., & MME., & DENIS (1916). Leishmaniose canine au Maroc. *Ibid.*, IX, 202.

DENIER (1907). Sur un piroplasme du *Cervus aristotelis* de l'Annam. *Ann. Inst. Pasteur*, XXI, 657.

DERRIEU & RAYNAUD, M. (1914). Dysenterie chronique à flagellé nouveau. *Bull. Soc. Path. Exot.*, VII, 571.

DESCHIENS, R. (1921). Les entérites à *Giardia* (*Lamblia*). *Trav. Lab. Parasit. Fac. Méd.*, Paris.
—— (1925). *Giardia cati* (n. sp.) du chat domestique. *C. R. Soc. Biol.*, XCII, 1271.
—— (1925a). Kystes de *Giardia* sp. observés chez le lion (*Felis leo*). *Ibid.*, XCIII, 1065.

DIAS, E. C., & ARAGÃO, H. DE B. (1914). Pesquizas sobre a natureza dos anaplasmas. *Mem. Inst. O. Cruz*, VI, 231.

DIEBEN, C. P. A. (1924). Over der morphologie en biologie van het ratten-coccidium *Eimeria nieschulzi* n. sp. en zijne verspreiding in Nederland. *Proefschr. Veeartsenijk. Hoogesch. Utrecht.*

DIESING, C. M. (1851). *Systema helminthium* (Vindobonæ). II, 15.

DIONISI, A. (1898). Un parassita del globulo rosso in una specie di pipistrello (*Miniopterus schreibersii* Kuhl). Nota preliminare. *Atti R. Accad. Lincei, Rendic.*, VII, ii, 214; Ein Parasit der roten Blutkörperchen in einer Fledermausart. Moleschott's *Unters. Nat. Mensch. Tiere.*, XVI, 531.
—— (1898a). I parassiti endoglobulari dei pipistrelli. *Atti. R. Accad. Lincei, Rendic.*, VII, ii, 254.
—— (1899). Les parasites endoglobulaires des chauves-souris. *Arch. Ital. Biol.*, XXXI, 153.
—— (1899a). La malaria di alcune specie di pipistrelli. *Atti Soc. Studi Malaria*, Rome, I, 133.
—— (1901). Die Malaria einiger Fledermausarten. Moleschott's *Unters. Nat. Mensch. Tiere.*, XVII, 281.

DISTASO, A. (1908). Sui processi vegetativi e sull' incistidamento di *Actinophrys sol*. *Arch. Protist.*, XII, 277.

DOBELL, C. (1907). Observations on the life-history of *Adelea ovata* Aimé Schneider, with a note on a new gregarine from the gut of *Lithobius forficatus*. *Proc. Roy. Soc.*, LXXIX, B, 155.
—— (1907a). *Trichomastix serpentis* n. sp. *Quart. Journ. Micro. Sci.*, LI, 449.
—— (1908). Some remarks upon the "autogamy" of *Bodo lacertæ* (Grassi). *Biol. Centralbl.*, XXVIII, 548.
—— (1908a). Some notes on the hæmogregarines parasitic in snakes. *Parasitology*, I, 288.
—— (1908b). The structure and life-history of *Copromonas subtilis* n. g., n. sp. (etc.). *Quart. Journ. Micro. Sci.*, LII, 75.
—— (1908c). On the intestinal Protozoan parasites of frogs and toads. *Proc. Cambridge Philos. Soc.*, XIV, 428.
—— (1909). Researches on the intestinal Protozoa of frogs and toads. *Quart. Journ. Micro. Sci.*, LIII, 201.
—— (1909a). The "autogamy" of *Bodo lacertæ*. A reply to Dr. v. Prowazek. *Biol. Centralbl.*, XXIX, 363.
—— (1910). On some parasitic Protozoa from Ceylon. *Spolia Zeylanica*, VII, 65.
—— (1910a). Contributions to the life-history of *Hæmocystidium simondi* Castellani et Willey. *Festschr. f. R. Hertwig*, Jena, I, 125.
—— (1911). The principles of protistology. *Arch. Protist.*, XXIII, 269.
—— (1914). Le cycle évolutif de l'*Aggregata*. Note préliminaire. *Bull. Inst. Océanogr.*, Monaco, No. 283, 1.
—— (1914a). Cytological studies on three species of *Amœba*—*A. lacertæ* Hartmann, *A. glebæ* n. sp., *A. fluvialis* n. sp. *Arch. Protist.*, XXXIV, 139.
—— (1917). Reports upon investigations in the United Kingdom of dysentery cases received from the Eastern Mediterranean. I. Amœbic dysentery and the protozoological investigation of cases and carriers. *Med. Res. Council, Spec. Rep. Series*, No. 4, London.
—— (1918). Are *Entamœba histolytica* and *Entamœba ranarum* the same species ? An experimental inquiry. *Parasitology*, X, 294.
—— (1919). *The Amœbæ living in man.* London.
—— (1919a). A revision of the *Coccidia* parasitic in man. *Parasitology*, XI, 147.
—— (1920). The discovery of the intestinal Protozoa of man. *Proc. Roy. Soc. Med.*, XIII (Sect. Hist. Med.), 1.
—— (1921). A report on the occurrence of intestinal Protozoa in the inhabitants of Britain, with special reference to *Entamœba histolytica*. *Med. Res. Council, Spec. Rep. Series*, No. 59, London.

DOBELL, C. (1921a). A note on the new species of *Eimeria* found in man by Dr. E. P. Snijders. *Parasitology*, XII, 433 (No. 4 published January, 1921).

—— (1922). The discovery of the *Coccidia*. *Ibid.*, XIV, 342.

—— (1925). The life-history and chromosome cycle of *Aggregata eberthi* (Protozoa, Sporozoa, Coccidia). *Ibid.*, XVII, 1.

—— (1926). [Amoebae of monkeys]. *Rept. Med. Research Council*. 1924-25, 31.

—— (1926a). On the species of *Isospora* parasitic in man. *Parasitology*, XVIII, 74.

DOBELL, C., GETTINGS, H. S., JEPPS, M. W., & STEPHENS. J. B. (1918). A study of 1,300 convalescent cases of dysentery from home hospitals, with special reference to the incidence and treatment of amœbic dysentery carriers. *Med. Res. Council, Spec. Rep. Series*, No. 15, London.

DOBELL, C., & JAMESON, A. P. (1915). The chromosome cycle in coccidia and gregarines. *Proc Roy. Soc.*, LXXXIX, 83.

DOBELL, C., & JEPPS, M. W. (1917). On the three common intestinal *Entamœbæ* of man and their differential diagnosis. *Brit. Med. Journ.*, I, 607.

—— (1918). A study of the diverse races of *Entamœba histolytica* distinguishable from one another by the dimensions of their cysts. *Parasitology*, X, 320.

DOBELL, C., & O'CONNOR, F. W. (1921). *The intestinal Protozoa of man*. London.

DOBELL, C., & STEVENSON, A. C. (1918). A note on the duration of infections with *Entamœba histolytica*. *Trans. Soc. Trop. Med. & Hyg.*, XI, 168.

DOCK, G. (1894). Flagellate Protozoa in freshly passed urine of a man; preliminary note. *Med. News*, Philadelphia, LXV, 690.

—— (1896). *Trichomonas* as a parasite of man. *Amer. Journ. Med. Sci.*, Philadelphia, CXI, 1.

DODD, S. (1912). *Trypanosoma ingens* in the mouse deer (*Tragulus javanicus*). *Journ. Comp. Path. & Therap.*, XXV, 281.

—— (1913). Anaplasms or Jolly bodies ? A contribution to the knowledge of certain intracorpuscular bodies present in the blood of some species of mammals. *Ibid.*, XXVI, 97.

DOERR, R., & ZDANSKY, E. (1923). Zur Aetiologie der Encephalitis epidemica. *Schweiz. Med. Woch.*, IV, 349.

—— —— (1923a). Weitere parasitologische Befunde im Gehirn von Kaninchen. *Ibid.*, IV, 1189.

—— —— (1923b). Parasitologische Befunde im Gehirne von Kaninchen, welche zu Encephalitisversuchen gedient hatten. *Zeitschr. Hyg. u. Infekt.*, CI, 239.

DOFLEIN, F. (1901). *Die Protozoen als Parasiten und Krankheitserreger*. Jena.

—— (1910). Studien zur Naturgeschichte der Protozoen. VI. Experimentelle Studien über die Trypanosomen des Frosches. *Arch. Protist.*, XIX, 207.

—— (1916). *Lehrbuch der Protozoenkunde*. 4th edition. Jena.

—— (1922). Untersuchungen über Chrysomonadinen. *Arch. Protist.*, XLIV, 149.

DOGIEL, V. (1923). *The transformation of the male pronucleus in a spermatozoon in certain Infusoria during their conjugation*. Petrograd.

—— (1925). Die Geschlechtsprozesse bei Infusorien (speziell bei den Ophryoscoleciden), neue Tatsachen und theoretische Erwägungen. *Arch. Protist.*, L, 283.

DONATIEN, A., & LESTOQUARD, F. (1923). Le *Debab* naturel du chien. Transmission par les stomoxes. *Bull. Soc. Path. Exot.*, XVI, 168.

DONATIEN, A., & PARROT, L. (1922). Trypanosomiase naturelle du chien au Sahara. *Ibid.*, XV, 549.

DONATIEN, A., PLANTUREUX, E., ROSSI, P., & ESPERANDIEU, G. (1923). La theilériose bovine en Algérie. *Ibid.*, XVI, 6.

DONIZIO, G. DI (1921). Su due tripanosomi del bestiame nella Somalia Italiana. *Ibid.*, XIV, 423.

DONNÉ, A. (1837). *Recherches microscopiques sur la nature des mucus et la matière des divers écoulements des organes génito-urinaires chez l'homme et chez la femme; description des nouveaux animalcules découverts dans quelques unes de ces fluides ; observations sur un nouveau mode de traitement de la blennorhagie*. Paris.

DONOVAN, C. (1903). The aetiology of one of the heterogeneous fevers in India. *Brit. Med. Journ.*, II, 1401.

—— (1905). Medical cases from Madras General Hospital. *Indian Med. Gaz.*, XL, 411.

—— (1909). Kala-azar. *Ann. Rept. & Statist. Govt. Gen. Hosp.*, Madras, *1908*, 28.

—— (1909a). Kala-azar in Madras. *Trans. Bombay Med. Congr.*, February *24*, 159.

—— (1909b). Kala-azar in Madras, especially with regard to its connection with the dog and the bug (*Conorrhinus*). *Lancet*, II, 1495.

—— (1913). Kala-azar, its distribution and the probable modes of infection. *Indian Journ. Med. Res.*, I, 177.

—— (1920). Malaria of monkeys. *Ibid.*, VII, 717.

DOUWES, J. B. (1921). Bijdrage tot de kennis van enkele darmprotozoen der huisdieren in het bijzonder bij schaap en varken. (Inaug. Diss.) Utrecht.

DOYLE, T. M. (1924). The occurrence of blue bodies as a developmental stage of *Theileria mutans*. *Journ. Comp. Path. & Therap.*, XXXVII, 18.

DRBOHLAV, J. J. (1924). The cultivation of the Protozoön of blackhead. *Journ. Med. Res.*, XLIV, 677.

—— (1925). [i.] Studies on the relation of insect herpetomonad and crithidial flagellates to leishmaniasis. I. Inoculation of mammals with insect flagellates. [ii.] Studies on the relation of insect herpetomonad flagellates to leishmaniasis. II. The specificity of the various insect flagellates for certain hosts as indicated by cross-infection experiments. [iii.] Studies on the relation of insect flagellates to leishmaniasis. III. Cultivation requirements. *Amer. Journ. Hyg.*, V, 580, 599, 611.

—— (1925a). Une nouvelle preuve de la possibilité de cultiver *Entamœba dysenteriæ* type *histolytica*. *Ann. Parasit. Hum. & Comp.*, III, 349.

—— (1925b). Culture d' *Entamœba dysenteriæ* type *Tetragena minuta*. *Ibid.*, III, 358.

—— (1925c). Culture d' *Entamœba gingivalis* (Gross, 1849), Brumpt, 1913. *Ibid.*, III, 361.

—— (1925d). Culture d' *Entamœba coli* Loesch, 1875, emend. Schaudinn, 1903. *Ibid.*, III, 364.

—— (1925e). Culture d' *Entamœba aulastomi* Nöller, 1919. *Ibid.*, III, 367.

—— (1925f). Culture de *Trichomonas sanguisugæ* Alexeieff, 1911. *Ibid.*, III, 369.

DREYER, W. (1910). Über durch Protozoen im Blut hervorgerufene Erkrankung bei Menschen und Tieren in Ägypten. *Arch. Schiffs.- u. Trop.-Hyg.*, XIV, 37.

DSCHUNKOWSKY, E., & LUHS, J. (1904). Die Piroplasmosen der Rinder. (Vorl. Mitt.) *Centralbl. Bakt.*, I Abt., XXXV, 486.

—— (1909). Entwickelungsformen von Piroplasmen in Zecken. 9e *Congr. Internat. Méd. Vét., La Haye.*

—— (1909a). Les maladies à protozoaires des animaux domestiques en Transcaucasie. Formes d'évolution des piroplasmes dans les tiques. *Ibid.*

—— —— (1909b). Leishmania beim Hunde in Transkaukasien. *Ibid.*

—— —— (1913). *Nuttallia* und *Piroplasma* bei der Piroplasmose der Einhufer in Transkaukasien. *Parasitology*, V, 289.

DSCHUNKOWSKY, E., & URODSCHEVICH, V. (1924). Theileriasis in goats, sheep, and cattle, with a description of *Theileria hirci* n. sp. from Serbia. *Ibid.*, XVI, 107.

DUBOSCQ, O. (1917). Sur un nouveau sporozoaire *Selysina perforans* n. g., n. sp. *C. R. Acad. Sci.*, CLXIV, 450.

—— (1918). *Selysina perforans* Dub. Description des stades connus du sporozoaire de *Stolonica* avec quelques remarques sur le pseudo-vitellus des statoblastes et sur les cellules géantes. *Arch. Zool. Exp.*, LVIII, 1.

DUBOSCQ, O., & GRASSÉ, P. (1923). Sur les petits flagellés de *Calotermes flavicollis* (Fab.). *C. R. Acad. Sci.*, CLXXVII, 362.

—— —— (1924). Notes sur les protistes parasites des termites de France. I. *Trichomonas trypanoides*, n. sp. *C. R. Soc. Biol.*, XC, 547.

—— —— (1924a). Notes sur les protistes parasites des termites de France. Sur l'évolution de *Janickiella grassii* Dub. et Gras. *Ibid.*, XC, 1442.

DUCLOUX, E. (1905). Sur une piroplasmose bacilliforme du bœuf en Tunisie. *Ibid.*, LIX, 461.

DUFOUR, L. (1828). Note sur la grégarine, nouveau genre de ver qui vit en troupeau dans les intestins de divers insectes. *Ann. Sci. Nat.*, XIII, 366.

DUJARDIN, F. (1841). *Histoire naturelle des zoophytes. Infusoires.* Paris.

DUKE, H. L. (1912). The transmission of *Trypanosoma nanum* Laveran. *Proc. Roy. Soc.*, LXXXV, B, 4.

—— (1912a). Antelope and their relation to trypanosomiasis. *Ibid.*, LXXXV, B, 156.

—— (1912b). Antelope as a reservoir for *Trypanosoma gambiense*. *Ibid.*, LXXXV, B, 299.

—— (1912c). Further observations on the recovery of *Trypanosoma gambiense* from *Tragelaphus spekei* on the islands of Lake Victoria Nyanza. *Ibid.*, LXXXV, B, 483.

—— (1913). A trypanosome from British East Africa, showing posterior-nuclear forms. *Rep. Sleep. Sickn. Comm. Roy. Soc.*, XIII. 67.

—— (1913a). Some trypanosomes recovered from wild game in Western Uganda. *Ibid.*, XIV, 37.

—— (1914). Wild game as a trypanosome reservoir in the Uganda Protectorate, with some criticisms on the current methods of diagnosing these Protozoa. *Arch. Protist.*, XXXII, 393.

—— (1915). The wild game and human trypanosomiasis, with some remarks on the nomenclature of certain Pan-African trypanosomes. *Journ. Trop. Med. & Hyg.*, XVIII, 13.

—— (1919). Tsetse flies and trypanosomiasis. Some questions suggested by the later history of the sleeping sickness epidemic in Uganda Protectorate. *Parasitology*, XI, 415.

—— (1921). On the zoological status of the polymorphic mammalian trypanosomes of Africa and their relation to man. *Ibid.*, XIII, 352.

—— (1923). An inquiry into an outbreak of human trypanosomiasis in a *Glossina morsitans* belt to the east of Mwanza, Tanganyika Territory. *Proc. Roy. Soc.*, XCIV, B, 250.

—— (1923a). Further inquiries into the zoological status of the polymorphic mammalian trypanosomes of Africa, and the means by which they are spread in nature. *Parasitology*, XV, 258.

DUKE, H. L. (1923b). *Annual Report on the Bacteriological Department for the year ended December 31, 1922*, Uganda Protectorate, 9.

—— (1923c). The transmission of *T. congolense* by *G. palpalis*. *Ann. Trop. Med. & Parasit.*, XVII, 511.

DUKE, H. L., & ROBERTSON, M. (1912). Observations on fowls and ducks in Uganda with relation to *Trypanosoma gallinarum* and *T. gambiense*. *Proc. Roy. Soc.*, LXXXV, B, 378.

DUNKERLY, J. S. (1911). On some stages in the life-history of *Leptomonas muscæ-domesticæ*, with some remarks on the relationships of the flagellate parasites of insects. *Quart. Journ. Micro. Sci.*, LVI, 645.

—— (1913). *Flagellata* and *Ciliata*. (Clare Island Survey.) *Proc. R. Irish Acad. Sci.*, XXXI, 1.

—— (1914). *Dermocystidium pusula* Pérez, parasitic on *Trutta fario*. *Zool. Anz.*, XLIV, 179.

—— (1921). Fish Myxosporidia from Plymouth. *Parasitology*, XII, 328. (Pt. 4 pub. January 10, 1921.)

—— (1921a). Nuclear division in the dinoflagellate, *Oxyrrhis marina*, Duj. *Proc. R. Phys. Soc. Edin.*, XX, 217.

—— (1921b). *Rhabdamœba marina*, gen. n., et sp. n. *Ibid.*, XX, 220.

—— (1925). The development and relationships of the Myxosporidia. *Quart. Journ. Micro. Sci.*, LXIX, 185.

DUPUY, A. (1888). Malaria des chevaux algériens en Sénégambie. *Rec. Méd. Vét.*, LXV, 535.

DURHAM, H. E. (1902). Drepanidium in the toad. *Liverpool Sch. Trop. Med.*, *Mem. VII*, 78.

—— (1908). Notes on nagana and on some Hæmatozoa observed during my travels. *Parasitology*, I, 227.

DUTTON, J. E. (1902). Preliminary note upon a trypanosome occurring in the blood of man. *Thomson Yates Lab. Repts.*, IV, 455.

DUTTON, J. E., & TODD, J. L. (1903). First report of the Trypanosomiasis Expedition to Senegambia (1902). *Liverpool Sch. Trop. Med.*, *Mem. XI*, 1.

DUTTON, J. E., TODD, J. L., & HANNINGTON, J. W. B. (1907). Trypanosome transmission experiments. *Ann. Trop. Med. & Parasit.*, I, 201.

DUTTON, J. E., TODD, J. L., & TOBEY, E. N. (1906). Concerning certain parasitic Protozoa observed in Africa. Part I. *Liverpool Sch. Trop. Med.*, *Mem. XXI*, 85.

—————— (1907). Concerning certain parasitic Protozoa observed in Africa. *Ann. Trop. Med. & Parasit.*, I, 287.

EBERLEIN, R. (1895). Über die im Wiederkäuermagen vorkommenden ciliaten Infusorien. *Zeitschr. Wiss. Zool.*, LIX, 233.

EDINGTON, A. (1908). Preliminary note on the occurrence of a new variety of trypanosomiasis on the island of Zanzibar. *Proc. Roy. Soc.*, LXXX, B, 545.

EDINGTON, A., & COUTTS, J. M. (1907). A note on a recent epidemic of trypanosomiasis at Mauritius. *Lancet*, II, 952.

EDWARDS, J. T. (1925). *Report of the Imperial Bacteriological Laboratory, Muktesar, for the two years ending March 31, 1924*. Calcutta. 50.

EECKE, J. VAN (1892). Sarcosporidien (Balbiani). *Geneesk. Tijdschr. Nederl.-Indië*, XXXII, 363.

EGGEBRECHT (1908). *Piroplasma canis*. *Zeitschr. Infektionskr. d. Haust.*, V, 129.

EHRENBERG, C. G. (1833). Dritter Beitrag zur Erkenntnis grosser Organisation in der Richtung des kleinsten Raumes. *Abh. K. Akad. Wiss. Berlin*, 145.

—— (1838). *Die Infusionsthierchen als vollkommene Organismen*. Leipzig.

EHRLICH, P., & SHIGA, K. (1904). Farbentherapeutische Versuche bei Trypanosomenerkrankung. *Berlin. Klin. Woch.*, XLI, 329, 362.

EICHHORN, A., & GALLAGHER, B. (1916). Spontaneous amoebic dysentery in monkeys. *Journ. Infect. Dis.*, XIX, 395.

EIMER, T. (1870). *Ueber die ei- und kugelförmigen sogenannten Psorospermien der Wirbelthiere.* Würzburg.

ELMASSIAN, M. (1909). Sur une nouvelle espèce amibienne chez l'homme, *Entamœba minuta*, n. sp., morphologie-évolution-pathogénie. *Centralbl. Bakt.*, I Abt., LII, 335.

ELMHIRST, R., & MARTIN, C. H. (1910). On a *Trypanoplasma* from the stomach of the conger eel (*Conger niger*). *Zool. Anz.*, XXXV, 475.

EMERY, C. (1909). I missosporidii sono Protozoi ? *Monit. Zool. Ital.*, XX, 247.

EMIN, A. (1914). Une variété nouvelle du parasite de Laveran. *Bull. Soc. Path. Exot.*, VII, 385.

ENGMAN, M. F., & HEITHAUS, A. S. (1919). Amebiasis cutis. *Journ. Cutan. Dis.*, XXXVII, 715.

ENRIQUES, P. (1903). Sulla così detta " degenerazione senile " dei Protozoi. *Monit. Zool. Ital.* XIV, 349.

—— (1907). La coniugazione e il differenziamento sessuale negli infusori. *Arch. Protist.*, IX, 195.

—— (1908). Sulla morfologia e sistematica del genere *Colpoda*. *Arch. Zool. Exp.*, VIII, (N. & R.), i.

—— (1908a). Die Conjugation und sexuelle Differenzierung der Infusorien. 2. Abhandlung: Wiederconjugante und Hemisexe bei *Chilodon*. *Arch. Protist.*, XII, 213.

ENRIQUES, P. (1916). Duemila cinquecento generazioni in un infusorio, senza coniugazione nè partenogenesi, nè depressioni. *Rend. Acc. Sci. Bologna*, N.S., XX, 67.

ENTZ, G. (1888). Über eine *Nyctotherus*art im Blute von *Apus cancriformis*. *Zool. Anz.*, XI, 618.

ENTZ, G., JUN. (1912). Über eine neue Amöbe auf Süsswasser-Polypen (*Hydra oligactis* Pall.). *Arch. Protist.*, XXVII, 19.

—— (1913). Cytologische Beobachtungen an *Polytoma uvella*. *Verhandl. Deutsch. Zool. Ges.*, XXIII, 249.

—— (1913*a*). Über Organisationsverhältnisse von *Nyctotherus piscicola* (Daday). *Arch. Protist.*, XXIX, 364.

—— (1918). Über die mitotische Teilung von *Polytoma uvella*. *Ibid.*, XXXVIII, 324.

EPSTEIN, A. (1893). Beobachtungen über *Monocercomonas hominis* (Grassi) und *Amœba coli* (Loesch) bei Kinder-Diarrhoen. *Prager Med. Woch.*, XVIII, 463, 475, 486.

EPSTEIN, H. V., & ILOVAISKI, S. A. (1914). Researches on parasitic amœbæ. (In Russian.) *Journ. Soc. Amis Sci. Nat., Anthrop., Ethnogr.* (Sect. Zool.), Moscow, N.S., II, No. 1.

ERDMANN, R. (1910). Die Entwicklung der *Sarcocystis muris* in der Muskulatur. *Sitzungsb. Ges. Naturf. Freunde*, VIII, 377.

—— (1910*a*). Beiträge zur Morphologie und Entwicklungsgeschichte des Hammelsarkosporids in der Maus. *Centralbl. Bakt.*, I. Abt., LIII, 510.

—— (1914). The schizogony in the life-cycle of *Sarcocystis muris*. *Proc. Soc. Exp. Biol. & Med.*, XI, 152.

—— (1917). New facts and views concerning the occurrence of a sexual process in the myxosporidian life-cycle. *Amer. Natural.*, LI, 719.

ESCOMEL, E. (1911). [Note on flagellate forms of *Leishmania* in human lesions in America.] *Gaceta Médica de Arequipa*, September 6.

—— (1913). Sur la dysentérie à *Trichomonas* à Arequipa (Pérou). *Bull. Soc. Path. Exot.*, VI, 120.

—— (1913*a*). Première découverte de *Leishmania tropica* flagellée dans le corps humain. *Ibid.*, VI, 237.

—— (1913*b*). Protozoarios de las aguas de Arequipa. El primer caso de lambliosis intestinal descrito en Péru. *Trab. 5° Congr. Méd. Lat. Amer.*, 6° Pan Amer. Lima.

—— (1914). Leishmania flagelada en el Peru. *Cron. Med.*, Lima, XXXI, 224.

—— (1919). La *Latrodectus mactans* ou "lucacha" au Pérou. Etude clinique et expérimentale de l'action du venin. *Bull. Soc. Path. Exot.*, XII, 702.

—— (1919*a*). La trypanosomiase humaine existe dans les forêts orientales du Pérou. *Ibid.*, XII, 723.

—— (1920). Comprobación clínica y microscópica de la existencia en el Perú de la tripanosomiasis americana. *An. Facul. Med. Lima*, V, 14.

—— (1922). *Leishmaniasis y blastomicosis en America.* Lima.

—— (1925). A propos de quelques flagellés de reptiles et de batraciens observés à Arequipa, Pérou. *Bull. Soc. Path. Exot.*, XVIII, 58.

EVANS, G. (1880). Report on "surra" disease in the Dera Ismail Khan district. *Punjab Govt. Milit. Dept.*, No. 493-4467.

EVANS, G. H. (1910). Elephant surra. *Journ. Trop. Vet. Sci.*, Calcutta, V, 233.

EVANS, G. H., & RENNIE, T. (1910). Trypanosomiasis in the elephant. *Ibid.*, V, 535.

FANO, C. DA (1924). Protozoan-like parasites in spontaneous encephalitis of rabbits. *Journ. Path. & Bact.*, XXVII, 333.

FANTHAM, H. B. (1905). *Lankesterella tritonis*, n. sp., a Hæmogregarine from the blood of the newt. *Triton cristatus* (*Molge cristata*). *Zool. Anz.*, XXIX, 257.

—— (1906). *Piroplasma muris* Fant. from the blood of the white rat, with remarks on the genus *Piroplasma*. *Quart. Journ. Micro. Sci.*, L, 493.

—— (1908). The schizogregarines: a review and a new classification. *Parasitology*, I, 369.

—— (1910). On the occurrence of schizogony in an avian *Leucocytozoon* (*L. lovati*) parasitic in the red grouse, *Lagopus scoticus*. *Ann. Trop. Med. & Parasit.*, IV, 255.

—— (1910*a*). The morphology and life-history of *Eimeria* (*Coccidium*) *avium*, a sporozoön causing a fatal disease among young grouse. *Proc. Zool. Soc.*, London, II, 672.

—— (1910*b*). Observations on the parasitic Protozoa of the red grouse (*Lagopus scoticus*) with a note on the grouse fly. *Ibid.*, II, 692.

—— (1911). The life-history of *Trypanosoma gambiense* and *Trypanosoma rhodesiense* as seen in rats and guinea-pigs. *Proc. Roy. Soc.*, LXXXIII, B, 212.

—— (1911*a*). On the amœbæ parasitic in the human intestine, with remarks on the life-cycle of *Entamœba coli* in cultures. *Ann. Trop. Med. & Parasit.*, V, 111.

—— (1912). Some insect flagellates and the problem of the transmission of Leishmania. *Brit. Med. Journ.*, II, 1196.

—— (1917). Some parasitic Protozoa of man and their probable evolution. *Med. Journ. S. Africa*, XIII, 33.

FANTHAM, H. B. (1919). Some parasitic Protozoa found in South Africa—II. *S. African Journ. Sci.*, XVI, 185.
—— (1920). Some parasitic Protozoa found in South Africa—III. *Ibid.*, XVII, 131.
—— (1921). Some parasitic Protozoa found in South Africa—IV. *Ibid.*, XVIII, 164.
—— (1922). Some parasitic Protozoa found in South Africa—V. *Ibid.*, XIX, 332.
—— (1923). Some parasitic Protozoa found in South Africa—VI. *Ibid.*, XX, 493.
—— (1924). Some parasitic Protozoa found in South Africa—VII. *Ibid.*, XXI, 435.
—— (1925). Some parasitic Protozoa found in South Africa—VIII. *Ibid.*, XXIII, 346
FANTHAM, H. B., & PORTER, A. (1910). On a new trypanoplasm, *T. dendrocœli*, sp. n., from *Dendrocœlum lacteum. Prcc. Zool. Soc.*, London, II, 670.
—— —— (1911). A bee disease due to a Protozoal parasite (*Nosema apis*). *Ibid.*, II, 625.
—— —— (1912). The morphology and life-history of *Nosema apis* and the significance of its various stages in the so-called " Isle of Wight " disease in bees (microsporidiosis). *Ann. Trop. Med. & Parasit.*, VI, 163.
—— —— (1915). On the natural occurrence of herpetomonads (leptomonads) in mice. *Parasitology*, VIII, 128.
—— —— (1915a). Some insect flagellates introduced into vertebrates. *Proc. Cambridge Philos. Soc.*, XVIII, 39.
—— —— (1915b). Further experimental researches on insect flagellates introduced into vertebrates. *Ibid.*, XVIII, 137.
—— —— (1915c). Some experimental researches on induced herpetomoniasis in birds. *Ann. Trop. Med. & Parasit.*, IX, 543.
—— —— (1916). The pathogenicity of *Giardia* (*Lamblia*) *intestinalis* to men and experimental animals. *Brit. Med. Journ.*, II, 139.
—— —— (1920). On the natural occurrence of herpetomonads (leptomonads) in the blood of a fish, *Dentex argyrozona*, and its significance. *Journ. Parasit.*, VII, 16.
FAUST, E. C. (1921). The human *Trichomonas* in North China. *Amer. Journ. Hyg.*, I, 410.
—— (1921a). A study of *Trichomonas* of the guinea-pig from Pekin. *Arch. Protist.*, XLIV, 115.
—— (1922). Notes on *Embadomonas sinensis* Faust and Wassell, 1921. *Journ. Parasit.*, IX, 33.
—— (1923). A new type of amœba parasitic in man observed in North China. *Ibid.*, IX, 221.
—— (1923a). The possible incidence of river fever (tsutsugamushi disease) in the central Yangtze valley. *China Med. Journ.*, XXXVII, 979.
FAUST, E. C., & WASSELL, C. McA. (1921). Preliminary survey of the intestinal parasites of man in the central Yangtze valley. *Ibid.*, XXXV, 532.
FEDOROVITCH, A. I. (1916). Hémoparasites trouvés dans un cas de fièvre chronique. *Ann. Inst. Pasteur*, XXX, 249.
FEHLANDT, O. (1911). Untersuchungen über Trypanosomen. (Inaug. Diss. Dok. Univ. Leipzig.)
FEIBEL, B. (1922). Die *Jodamœba butschlii* beim Schweine und die Bedeutung dieser Infektion des Schweines für den Befall des Menschen mit diesen Parasiten. (Inaug. Diss.) Hamburg.
FELETTI, R., & GRASSI, B. (1890). Sui parassiti della malaria. *Riforma Med.*, Naples, XI, 62. (French transl. *Arch. Ital. Biol.*, XIII, 287.).
FERMOR, X. (1913). Die Bedeutung der Encystierung bei *Stylonychia pustulata* Ehrbg. *Zool. Anz.*, XLII, 380.
FINCK, H. (1854). Sur la physiologie de l'épithélium intestinal. (Thèse.) Strasbourg. (Extract in Davaine's *Traité des Entozoaires*, 1860.)
FINKELSTEIN, N. J. (1908). Parasites endoglobulaires du sang chez les animaux à sang froid du Caucase. *Arch. Sci. Biol.*, St. Petersburg, XIII, 137.
FIORENTINI, A. (1890). *Intorno ai Protisti dell' intestino degli equini.* Pavia.
FIRTH, R. H. (1891). Notes on the appearance of certain sporozoöid bodies in the protoplasm of an " oriental sore." *Brit. Med. Journ.*, I, 60.
FISCHER, W. (1918). Amœbæ and dysentery. *China Med. Journ.*, XXXII, 13.
FLESCH, M. (1883). Über ein Sporozoon beim Pferde. *Zool. Anz.*, VI, 396.
—— (1884). Sur un parasite de la paroi intestinale du cheval. *Recueil Zool. Suisse*, I, 459.
—— (1884a). Über einen Parasiten in der Darmwand des Pferdes. *Mitt. Naturf. Ges. Bern*, 26.
FLU, P. C. (1908). Über die Flagellaten im Darm von *Melophagus ovinus*, *Arch. Protist.*, XII, 147.
—— (1908a). Untersuchungen über Affenmalaria. *Ibid.*, XII, 323.
—— (1911). Studien über die im Darm der Stubenfliege, *Musca domestica*, vorkommenden protozoären Gebilde. *Centralbl. Bakt.*, I. Abt., LVII, 522.
FOÀ, A. (1904). Ricerche intorno a due specie di flagellati parassiti. *Atti R. Accad. Lincei, Rendic.*, XIII, i, 121.
—— (1905). Due nuovi flagellati parassiti. *Ibid.*, XIV, 542.
FOLEY, H., & CATANEI, A. (1925). L'hémogrégarine de la vipère à cornes (*Hæmogregarina seurati*, Laveran et Pettit, 1911); ses formes de multiplication. *Bull. Soc. Path. Exot.*, XVIII, 393.
FONSECA, O. O. R., DA (1915). Sobre os flagellados dos mammiferos do Brazil. Um novo parasito do homen *Brazil-Medico*, XXIX, 281.

FONSECA, O. O. R. DA (1916). Estudos sobre os flagellados parasitos dos mammiferos do Brazil. *Mem. Inst. O. Cruz*, VIII, 5.

—— (1917). Sobre os flagellados parasitos (5ª nota prévia). *Brazil-Medico*, XXXI, 417.

—— (1918). Sobre os flagellados parasitos. Infeccões por *Enteromonas* (9ª nota prévia). *Ibid.*, XXXII, 313.

—— (1920). Estudos sobre os flagellados parasitos. *Mem. Inst. O. Cruz*, XII, 51.

FORDE, R. M. (1902). Some clinical notes on a European patient in whose blood a trypanosome was observed. *Journ. Trop. Med. & Hyg.*, V, 261.

FOULON, C. (1919). *Merocystis kathœ* Dakin. Une aggrégate de *Buccinum undatum*. *La Cellule*, XXX, 123.

FOX, E. C. R., & MACKIE, F. P. (1921). The formol-gel test in kala-azar. *Indian Med. Gaz.*, LVI, 374.

FRAENKEL, E. (1914). Ueber Fleckfieber und Roseola. *Munch. Med. Woch.*, LXI, 57.

FRANÇA, C. (1908). Sur une piroplasmose nouvelle chez une mangouste. *Bull. Soc. Path. Exot.*, I, 410.

—— (1908a). Le cycle évolutif des trypanosomes de la grenouille (*T. costatum, T. rotatorum* et *T. inopinatum*). *Arq. R. Inst. Bact., Camara Pestana*, II, 89.

—— (1908b). Une hémogregarine de l'anguille. *Ibid.*, II, 109.

—— (1908c). Quelques notes sur l'*Hœmogregarina splendens* Labbé. *Ibid.*, II, 123.

—— (1908d). Encore sur la trypanosome de *Hyla arborea*. *Ibid.*, II, 271.

—— (1909). Le cycle évolutif des trypanosomes de la grenouille. Remarques à propos du travail de MM. W. S. Patton et C. Strickland. *Ibid.*, II, 381.

—— (1910). Sur la classification des piroplasmes et description de deux formes de ces parasites. *Ibid.*, III, 11.

—— (1911). Quelques notes sur *Leptomonas davidi* Lafont. *Bull. Soc. Path. Exot.*, IV, 669.

—— (1911a). Notes sur les hématozoaires de la Guinée Portugaise. *Arq. R. Inst. Bact., Camara Pestana*, III, 229.

—— (1911b). Sur la relation autogénétique entre les grands et les petits trypanosomes de la grenouille. *C. R. Soc. Biol.*, LXX, 978.

—— (1912). Contribution à l'étude des *Leucocytozoon* des oiseaux du Portugal. *Bull. Soc. Path. Exot.*, V, 82, 173.

—— (1912a). Quelques considérations sur le genre *Theileria* et description d'une nouvelle espèce de ce genre (*Theileria stordii*). *Centralbl. Bakt.*, I. Abt., LXVII, 171.

—— (1912b). Sur les hématozoaires des taupes. *Arq. R. Inst. Bact., Camara Pestana*, III, 271.

—— (1913). Quelques observations sur les genres *Monocercomonas* et *Polymastix*. *Bull. Soc. Portugaise Sci. Nat.*, VI, 105.

—— (1914). La flagellose des euphorbes. *Arch. Protist.*, XXXIV, 108.

—— (1915). Le *Trypanosoma inopinatum*. *Ibid.*, XXXVI, 1.

—— (1917). Sur la classification des hémosporidies. *Jorn. Sci. Math. Fis. e Nat. Accad. Sci.*, Lisbon, I, 1.

—— (1918). Quelques considérations sur la classification des hématozoaires. *Ibid.*, I, 221.

—— (1918a). Sur un piroplasmide des bovidés de la Côte d'Or (*Achromaticus macfiei*, n. sp.). *An. Sci. d. Fac. Med. Porto*, IV, No. 3.

—— (1919). L'insecte transmetteur de *Leptomonas davidi*. (Note préliminaire.) *Bull. Soc. Path. Exot.*, XII, 513.

—— (1920). Quelques considérations sur la pathogénéité. *Rivista Biol.*, Rome, II, 273.

—— (1920a). La flagellose des euphorbes—II. *Ann. Inst. Pasteur*, XXXIV, 432.

—— (1921). Sur deux phytoflagellés (*L. elmassiani* Migone et *L. bordasi*, sp. n.). *Ann. Soc Belge Méd. Trop.*, I, 245.

—— (1922). Encore quelques considérations sur la flagellose des euphorbes. *Bull. Soc. Path. Exot.*, XV, 166.

—— (1925). Notes parasitologiques sur l'Angola. *Ann. Parasit. Hum. & Comp.*, III, 255.

FRANÇA, C., & ATHIAS, M. (1906). Notes cytologiques sur les trypanosomes parasites de la grenouille (*Rana esculenta*). *XVe Congr. Internat. Méd.*, Sect. I., Anat., Lisbon, 394.

—— —— (1907). Recherches sur les trypanosomes des amphibiens. II. Le *Trypanosoma rotatorium* de *Hyla arborea*. *Arq. R. Inst. Bact., Camara Pestana*, I, 289.

FRANCA, C., & PINTO, M. (1911). Sur le leucocytozoaire du *Mus decumanus*. *Ibid.*, III, 207.

FRANCHINI, F. (1920). Dissenteria amebica spontanea nel gatto concomitante ad una circonscritta epidemia di dissenteria nell' uomo in un comune del bolognese. (Nota preventiva.) *Giorn. Clin. Med.*, I, 352.

—— (1921). Amebiasi nella Provincia di Bologna. I. Dissenteria amebica nell' uomo. *Pathologica*, XIII, 529.

—— (1923). Amebiasi spontanea nel gatto concomitante ad una epidemia di dissenteria amebica nell' uomo. *Ann. Med. Nav.*, XXIX, i, 1.

FRANCHINI, G. (1911). Infezione sperimentale nella cavia da *Leishmania donovani*. *Malaria e Mal. Paesi Caldi*, II, 98.

FRANCHINI, G. (1911a). La *Leishmania donovani* può vivere e svilupparsi nel tubo digerente dell' *Anopheles cl[aviger]*. *Ibid.*, II, 324.

—— (1911b). La vita e lo sviluppo della *Leishmania donovani* nelle cimici, nelle pulci e nei pidocchi. *Studi Mal. Trop. Ital. Merid.*, II, 1.

—— (1912). On the presence of *Leishmania* in the digestive tract of *Anopheles maculipennis*. *Ann. Trop. Med. & Parasit.*, VI, 41.

—— (1913). Un nouveau protozoaire parasite de l'homme provenant du Brésil. *Bull. Soc. Path. Exot.*, VI, 156.

—— (1921). Trypanosome de la chauve-souris en Italie. Formes viscérales et stades de développement chez un acarien gamaside, le *Leiognathus laverani*, n. sp. *Ibid.*, XIV, 542.

—— (1921a). Sur les flagellés intestinaux du type *Herpetomonas* du *Chameleon vulgaris* et leur culture, et sur les flagellés du type *Herpetomonas* de *Chalcides (Gongylus) ocellatus* et *Tarentola mauritanica*. *Ibid.*, XIV, 641.

—— (1921b). Nuove ricerche su piante a lattice, in ispecie Apocinee ed Asclepiadee. (Nota preventiva.) *Pathologica*, XIII, 474.

—— (1922). Essais d'inoculation aux souris blanches du latex parasité de différentes espèces d'euphorbes. *Ann. Inst. Pasteur*, XXXVI, 873.

—— (1922a). *Hæmoproteus chelidonis* chez l'hirondelle *Chelidon urbica* Linn. Stades probables de développement chez la puce *Ceratophyllus hirundinis*. *Bull. Soc. Path. Exot.*, XV, 13.

—— (1922b). Sur un trypanosome du latex de deux espèces d'euphorbes. *Ibid.*, XV, 18.

—— (1922c). Sur un flagellé nouveau du latex de deux apocynées. *Ibid.*, XV, 109.

—— (1922d). Sur un flagellé de Lygœide (*Crithidia oxycareni*, n. sp.). *Ibid.*, XV, 113.

—— (1922e). Au sujet de la culture de l'*Herpetomonas* du *Pyrrhocoris apterus*. *Ibid.*, XV, 161.

—— (1922f). Flagellose du chou et des punaises du chou. *Ibid.*, XV, 163.

—— (1922g). Amibes et autres protozoaires de plantes à latex du Muséum de Paris. (Note préliminaire.) *Ibid.*, XV, 197.

—— (1922h). Sur une amibe des figuiers de plein air de la région parisienne et sa culture. *Ibid.*, XV, 287.

—— (1922i). Nouvelles recherches sur les trypanosomes des euphorbes et sur leur culture. *Ibid.*, XV, 299.

—— (1922j). Sur une amibe particulière d'une asclépiadacée (*Chlorocodon whitei*). *Ibid.*, XV, 393.

—— (1922k). Flagellés et amibes d'une articacée exotique—*Ficus parietalis*. *Ibid.*, XV, 399.

—— (1922l). Sur une amibe de la laitue (*Lactuca sativa*). *Ibid.*, XV, 784.

—— (1922m). Essais d'inoculation de différents protozoaires dans le latex des euphorbes. *Ibid.*, XV, 792.

—— (1922n). Essais d'inoculation au chat, d'amibes du latex de plantes. *Ibid.*, XV, 931.

—— (1922o). Protozoaires de muscides divers capturés sur des euphorbes. *Ibid.*, XV, 970.

—— (1922p). Sulla presenza dell'*Herpetomonas* (*Leptomonas*) *davidi* nel tubo digestivo de mosche catturate su della euforbie. (Nota preventiva.) *Pathologica*, XIV, 568.

—— (1923). Essai d'inoculation de latex parasités aux souris blanches. Abcès du foie expérimentaux déterminés par les amibes des latex. *Bull. Soc. Path. Exot.*, XVI, 162.

—— (1923a). Inoculation de flagellés d'insectes dans le latex des euphorbes. *Ibid.*, XVI, 646.

—— (1924). Observations sur les hématozoaires des oiseaux d'Italie. *Ann. Inst. Pasteur*, XXXVIII, 470.

—— (1924a). Piroplasmose chez un petit loir (*Myoxus avellanarius*). *Bull. Soc. Path. Exot.*, XVII, 881.

FRANCHINI, G., & MANTOVANI, M. (1913). Il primo caso di leishmaniosi cutanea brasiliana in Italia diagnosticato col reperto del parassita. *Pathologica*, V, 163.

—— —— (1915). Infection expérimentale du rat et de la souris par *Herpetomonas muscæ domesticæ*. *Bull. Soc. Path. Exot.*, VIII, 109.

FRANCHINI, G., & SAINI, M. (1923). Sur la présence d'hémogrégarines chez les poissons d'eau douce de France. *Ibid.*, XVI, 353.

FRANCO, E., & BORGES, J. (1916). Sur la sarcosporidiose bovine. *Arq. R. Inst. Bact., Camara Pestana*, IV, 269.

FRANKE, E. (1905). Therapeutische Versuche bei Trypanosomenerkrankung. (Diss., Giessen). Jena.

FRASER, A. D., & DUKE, H. L. (1912). Antelope infected with *Trypanosoma gambiense*. *Proc. Roy. Soc.*, LXXXIV, B, 484; *Rep. Sleep. Sickn. Comm. Roy. Soc.*, XIV, 60.

—— —— (1912a). An antelope trypanosome. The relation of wild animals to trypanosomiasis. *Proc. Roy. Soc.*, LXXXV, B, 1, 2.

—— —— (1912b). Various experiments. *Rep. Sleep. Sickn. Comm. Roy. Soc.*, XII, 75.

—— —— (1912c). The development of trypanosomes in *Glossina palpalis*. *Ibid.*, XII, 36.

FRASER, H., & SYMONDS, S. L. (1908). Surra in the Federated Malay States. *Studies Inst. Med. Res., F.M.S.*, No. 9.

FRENZEL, J. (1885). Über einige in Seethieren lebende Gregarinen. *Arch. Mikr. Anat.*, XXIV, 545.

FRENZEL. J. (1885a). Über den Darmkanal der Crustaceen nebst Bemerkungen zur Epithel-regeneration. *Ibid.*, XXV, 137.

—— (1892). Untersuchungen über die mikroskopische Fauna Argentiniens. I. & II. Abt.: Die Rhizopoden und Helioamœben. *Bibl. Zool.*, Stuttgart, Heft 12.

FRIEDRICH, L. (1909). Ueber Bau und Naturgeschichte des *Trypanoplasma helicis* (Leidy). *Arch. Protist.*, XIV, 363.

FROMENTEL, E. DE (1874). *Etudes sur les microzoaires ou infusoires proprement dits.* Paris.

FROSCH, P. (1909). Ätiologische Ermittelungen über das *Trypanosoma Frank. Zeitschr. Infektionskr. d. Haust.*, V, 316.

GACHET (1915). Thérapeutique spécifique et prophylaxie du bouton d'orient. *Bull. Acad. Méd.*, LXXIII, 475.

GÄBEL, M. (1914). Zur Pathogenität der Flagellaten. Ein Fall von Tetramitidendiarrhoe. *Arch. Protist.*, XXXIV, 1.

GALLAGHER, G. H. (1913). [Trypanosomes.] *Ann. Med. Rept.*, 1912. S. Nigeria, 7, 22.

—— (1914). The transmission of *Trypanosoma brucei* of Nigeria by *Glossina tachinoides*, with some notes on *Trypanosoma nigeriense. Journ. Trop. Med. & Hyg.*, XVII, 372.

GALLIARD & BRUMPT (1912). Un cas de dysentérie amibienne autochtone. *Bull. & Mém. Soc . Méd. Hôpit. Paris*, XXXIV, 736.

GALLI-VALERIO, B. (1903). Notes de parasitologie. *Centralbl. Bakt.*, I. Abt., XXXV, 81.

—— (1905). Notes de parasitologie et de technique parasitologique. *Ibid.*, I. Abt., XXXIX, 230.

—— (1905a). Einige Parasiten von *Arvicola nivalis. Zool. Anz.*, XXVIII, 519.

—— (1907). Notes médicales sur la Tunisie. *Bull. Soc. Vaudoise Sci. Nat.*, XLIII, 201.

—— (1911). Sur un *Piroplasma* d'*Erinaceus algirus. Centralbl. Bakt.*, I. Abt., LVIII, 565.

—— (1914). *Smithia talpœ*, n. sp. (*Piroplasmidœ*), chez *Talpa europœa* L. *Ibid.*, I Abt., LXXIII, 142.

—— (1921). La flagelliase des euphorbiacées en Suisse. *Schweiz. Med. Woch.*, II, 1154.

—— (1922). Parasitologische Untersuchungen und Beiträge zur parasitologischen Technik. *Centralbl. Bakt.*, II. Abt., LVI, 344.

—— (1923). Parasitologische Untersuchungen und Beiträge zur parasitologischen Technik. *Ibid.*, I. Abt., XCI, 120.

GASSOWSKY, G. (1919). Über die Mikrofauna des Pferdedarms. *Trav. Soc. Nat. Petrograd* (1918), XLIX, 20.

GATENBY, J B., & KING. S. D. (1925). *Opalina ranarum :* a flagellate. *Nature*, CXVI, 712.

GAUDUCHEAU, A. (1907). Culture d'une amibe dysentérique. *Gaz. Hebd. Sci. Méd. Bordeaux*, XXVIII, 486.

—— (1908). Formation de corps spirillaires dans une culture d'amibe. *C. R. Soc. Biol.*, LXIV, 493.

—— (1922). Remarques sur les amibes cultivables parasites de l'intestin de l'homme. *Bull. Soc. Path. Exot.*, XV, 229.

GAUTHIER, M. (1920). Présence d'un infusoire parasite dans l'estomac d'un saumon de fontaine. *C. R. Soc. Biol.*, LXXXIII, 1607.

GEISLER (1912). Trypanosomen beim ostafrikanischen Warzenschwein. *Arch. Schiffs.- u. Trop.-Hyg.*, XVI, 197.

GELEI, J. (1913). Bau. Teilung und Infektionsverhältnisse vom *Trypanoplasma dendrocœli* Fantham. *Arch. Protist.*, XXXII, 171.

GEORGÉVITCH, J. (1917). Esquisses protistologiques. III. Sur le cycle évolutif de *Ceratomyxa coris* Georgévitch. *Bull. Inst. Océanogr.*, Monaco, No. 328.

GÉRARD, P. (1913). Le cycle évolutif d'une nouvelle coccidie aviaire, *Eimeria bracheti* (n. sp.). (*Pfeifferia avium* Labbe (?), *Eimeria avium* Hadley.) *Arch. Protist.*, XXIX, 193.

GERRARD. P. N. (1906). On a Protozoan parasite found in the polymorphonuclear leucocytes of a dog. *Journ. Hyg.*, VI, 229.

GHOSH, E. (1922). On a new ciliate, *Balantidium blattarum*, sp. nov., intestinal parasite in the common cockroach (*Blatta americana*). *Parasitology*, XIV, 15.

—— (1922a). On a new ciliate, *Balantidium ovatum*, sp. nov., an intestinal parasite in the common cockroach (*Blatta americana*). *Ibid.*, XIV, 371.

—— (1925). On a new ciliate, *Balantidium knowlesii*, sp. nov., a cœlomic parasite in *Culicoides peregrinus. Ibid.*, XVII, 189.

GIARD, A. (1884). Note sur un nouveau groupe de protozoaires parasites des annélides. Sur quelques points de l'histoire des grégarines (*Selenidium pendula*). *C. R. Assoc. Franc. Avanc. Sci.*, 192.

GILL, C. A. (1921). The influence of humidity on the life-history of mosquitoes and on their power to transmit infection. *Trans. Roy. Soc. Trop. Med. & Hyg.*, XIV, 77.

GILRUTH. J. A. (1910). Notes on a Protozoon parasite found in the mucous membrane of the abomasum of a sheep. *Bull. Soc. Path. Exot.*, III, 297.

GILRUTH, J. A., & BULL, L. B. (1912).   Enteritis, associated with infection of the intestinal wall by cyst-forming Protozoa (Neosporidia), occurring in certain native animals (wallaby, kangaroo, and wombat). *Proc. Roy. Soc. Victoria*, XXIV, 432.

GILRUTH, J. A., SWEET, G., & DODD, S. (1910).   Notes on blood parasites. *Ibid.*, XXIII, 231.

GIUGNI, F. (1914).   La emocultura della *Leishmania donovani* dal sangue periferico in un caso di kala-azar nel bacino del Mediterraneo. *Pathologica*, VI. 284.

—— (1914a).   Due nuovi casi di reperto colturale della *Leishmania tropica* del bottone d'oriente. *Malaria e Mal. Paesi Caldi*, V, 183.

GLÄSER, H. (1912).   Untersuchungen über die Teilung einiger Amöben, zugleich ein Beitrag zur Phylogenie des Centrosomes. *Arch. Protist.*, XXV. 27.

GLASER, R. W. (1922).   *Herpetomonas muscæ domesticæ*, its behaviour and effect in laboratory animals. *Journ. Parasit.*, VIII, 99.

—— (1922a).   A study of *Trypanosoma americanum*. *Ibid.*, VIII, 136.

GLASGOW, H. (1914).   The gastric cæca and the cæcal bacteria of *Heteroptera*. *Biol. Bull. Marine Biol. Lab. Woods Hole, Mass.*, XXVI, 101.

GLUGE, G. (1838).   Notice sur quelques points d'anatomie pathologique comparée suivie de quelques observations sur la structure des branchies dans les epinoches. *Bull Acad. R. Belgique*, V. 771.

—— (1842).   Ueber ein eigenthümliches Entozoon im Blute des Frosches. *Arch. Anat. Phys. Wiss. Med.*, 148.

GODOY, A.. & PINTO, C. (1922).   *Caulleryella maligna*, n. sp.   Schizogregarina pathogenica para *Cellia allopha* Lutz e Peryassu. (Nota prévia.) *Brazil-Medico*, XXXVI, i. 46.

GOLDSCHMIDT, R. (1907).   Lebensgeschichte der Mastigamöben *Mastigella vitrea*. n. sp., und *Mastigina setosa*. n. sp   *Arch. Protist.*, Supplt. I, 83.

GOLGI, C. (1886).   Sull' infezione malarica. [Letter to Professors Marchiafava and Celli.] *Arch. Sci. Med.*, X, 109.

—— (1886a).   Ancora sulla infezione malarica. *Gaz. Ospid.*, VII, 419.

—— (1889).   Sul ciclo evolutivo dei parassiti malarici nella febbre terzana; diagnosi differenziale tra i parassiti endoglobulari malarici della terzana e quelli della quartana. *Arch. Sci. Med.*, XIII. 173.

GONDER, R. (1906).   *Achromaticus vesperuginis* (Dionisi). *Arb. K. Gesundheitsamte*, XXIV, 220.

—— (1910).   *Trypanosoma vespertilionis* (Battaglia). *Centralbl. Bakt.*, I. Abt., LIII, 293.

—— (1910a).   Die Entwicklung von *Theileria parva*, dem Erreger des Küstenfiebers der Rinder in Afrika. *Arch. Protist.*, XXI, 143.

—— (1910b).   *Lamblia sanguinis*, n. sp. (Gonder). *Ibid.*, XXI. 209.

—— (1911).   The development of *Theileria parva*, the cause of East Coast fever of cattle in South Africa. *Rept. Govt. Vet. Bact.*, 1909-10. 69.

—— (1911a).   *Theileria parva* und *Babesia mutans*, Küstenfieberparasit und Pseudoküstenfieberparasit. *Arch. Protist.*, XXI, 222.

—— (1911b).   Die Entwicklung von *Theileria parva*, dem Erreger des Küstenfiebers der Rinder in Afrika—II. *Ibid.*, XXII, 170.

—— (1913).   Experimentelle Uebertragung von Orientbeule auf Mäuse. *Arch. Schiffs.- u. Trop.-Hyg.*, XVII, 397.

—— (1915).   Zur Übertragung von *Hæmoproteus columbæ*. *Arch. Protist.*, XXXV, 316.

GONDER, R., & BERENBERG-GOSSLER, H. (1908).   Untersuchungen über Malariaplasmodien der Affen. *Malaria*, I, 47.

GONDER, R., & RODENWALDT, E. (1910).   Experimentelle Untersuchungen über Affenmalaria. *Centralbl. Bakt.*, I. Abt., LIV, 236.

GONZALEZ-MARTINEZ, I. (1920).   Les méthodes de laboratoire dans le diagnostic de la dairrhée chronique des pays chauds. *Bull. Soc. Path. Exot.*, XIII, 384.

GOODEY, T., & WELLINGS, A. W. (1917).   Observations on *Entamœba gingivalis* from the human mouth, with a note on the trichomonad flagellate, *Tetratrichomonas buccalis*, n. sp. *Parasitology*, IX. 537.

GOODPASTURE, E. W. (1924).   Spontaneous encephalitis in rabbits. *Journ. Infect. Dis.*, XXXIV, 428.

GOODRICH, H. P.. & MOSELEY, M. (1916).   On certain parasites of the mouth in cases of pyorrhœa. (Preliminary communication.) *Journ. R. Micro. Soc.*, 513.

GRAHAM-SMITH, G. S. (1905).   A new form of parasite found in the red blood-corpuscles of moles. *Journ. Hyg.*, V, 453.

GRANATA, L. (1914).   Ricerche sul ciclo evolutivo di *Haplosporidium limnodrili* Granata. *Arch. Protist.*, XXXV, 47.

—— (1925).   Gli attinomissidi. *Ibid.*, L, 139.

GRASSÉ, P. P. (1924).   *Octomastix parvus* Alex.   Diplozoaire parasite de la cistude d'Europe. *C. R. Soc. Biol.*, XCI, 439.

GRASSI, B. (1879).   Intorno a speciali corpuscoli (psorospermi) dell' uomo. *Rendic. R. Ist. Lomb. Sci.*, Milan, XII (2), 632.

GRASSI, B. (1879a). Dei Protozoi parassiti e specialmente di quelli che sono nell' uomo. *Gaz Med. Ital. Lombard.*, XXXIX, 445.

—— (1881). Di un nuovo parassita dell' uomo, *Megastoma entericum. Gaz. Ospid.*, II, 577.

—— (1881a). Intorno ad alcuni protisti endoparassitici, ed appartenenti alle classi dei Flagellati, Lobosi, Sporozoi e Ciliati. *Atti Soc. Ital. Sci. Nat.*, XXIV, 135. (French version. *Arch. Ital. Biol.*, II, 402: III, 23.)

—— (1888). Morfologia e sistematica di alcuni protozoi parassiti. *Atti R. Accad. Lincei, Rendic.*, 4 ser., IV, i, 5.

—— (1888a). Significato patologico dei protozoi parassiti dell' uomo. *Ibid.*, IV, i, 83.

—— (1899). Ancora sulla malaria. (Nota preliminare.) *Ibid.*, 5 ser., VIII, i, 559.

—— (1900). *Studi di uno zoologo sulla malaria.* (Pub. June 4, 1900.) Rome. (German version: *Die Malaria.* Jena, 1901.)

—— (1917). Flagellati viventi nei termiti. *Mem. R. Accad. Lincei*, 5 ser., XII, 331.

—— (1919). L'expérience de prophylaxie antimalarique à Fiumicino. *Bull. Off. Inter. Hyg. Pub.*, XI, 592.

GRASSI, B., BIGNAMI, A., & BASTIANELLI, G. (1898). Ulteriori ricerche sul ciclo dei parassiti malarici umani nel corpo del zanzarone. (Nota preliminare.) *Atti. R. Accad. Lincei, Rendic*, 5 ser., VIII, i, 21.

—— —— (1899). Resoconto degli studi fatti sulla malaria durante il mese di gennaio *Ibid.*, VIII, i. 100.

—— —— —— (1899a). Ulteriori ricerche sulla malaria. *Ibid.*, VIII, i, 434.

GRASSI, B. & FELETTI, R. (1890). Parassiti malarici negli uccelli. *Boll. mens. Accad. Giœnia Sci. Nat. Catania*, XIII, 3; XIV, 2 (Transl. *Arch. Ital. Biol.*, 1890, XIII, 297; *Centralbl. Bakt.*, 1890, I. Abt., VII. 396).

—— —— (1891). Weiteres zur Malariafrage. *Centralbl. Bakt.*, I. Abt., X, 449, 481, 517.

—— —— (1892). Contribuzione allo studio dei parassiti malarici. *Atti Accad. Giœnia Sci. Nat. Catania*, 4 ser., V. 1.

GRASSI, B., & SCHEWIAKOFF, W. (1888). Beitrag zur Kenntnis des *Megastoma entericum. Zeitschr. Wiss. Zool.*, XLVI. 143.

GRASSI, B., & SELLA, M. (1920). Seconda relazione della lotta antimalarica a Fiumicino (Roma). *Annali d'Igiene*, XXX, Supplt.

GRAY, A. C. H. (1913). Leishmaniose naturelle du chien à Tunis. *Bull. Soc. Path. Exot.*, VI, 165.

GRAY, A. C. H., & TULLOCH, F. M. G. (1905). The multiplication of *Trypanosoma gambiense* in the alimentary canal of *Glossina palpalis. Rep. Sleep. Sickn. Comm. Roy. Soc.*, VI. 282.

—— —— (1907). Continuation report on sleeping sickness in Uganda. *Ibid.*, VIII, 3.

GREIG, E. D. W., & GRAY, A. C. H. (1904). Note on the lymphatic glands in sleeping sickness. *Brit. Med. Journ.*, I, 1252.

GREINER, J. (1918). Cytologische Untersuchungen bei den Gametenbildung und Befruchtung des Coccids *Adelea ovata. Biol. Centralbl.*, XXXVIII, 522.

GRIESSMANN, K. (1913). Über marine Flagellaten. *Arch. Protist.*, XXXII, 1.

GRIFFITHS, J. A. (1918). A note on piroplasmosis of the donkey. *Journ. Comp. Path. & Therap.*, XXXI. 131.

GROS, G. (1849). Fragments d'helminthologie et de physiologie microscopique. *Bull. Soc. Imp. Nat. Moscou*, XXII, 549.

GRUBY, D. (1843). Recherches et observations sur une nouvelle espèce d'hématozoaire, *Trypanosoma sanguinis. C. R. Acad. Sci.*, XVII, 1134.

GUALDI, T., & MARTIRANO, F. (1901). L'azione della chinina sulle semilune. *Atti. Soc. Studi d. Malaria. Roma.* II, 1.

GUBLER, A. (1858). Tumeurs du foie déterminées par des œufs d'helminthes et comparables à des galles, observées chez l'homme. *C. R. Soc. Biol.*, V. 61; *Gaz. Méd. Paris*, XIII, 657.

GÜNTHER, K. (1914). Ueber eine Gregarine in *Ficalbia dofleini* Günther. *Zool. Anz.*, XLIV, 264.

GUÉRIN, F. (1926). Infection par *Nyctotherus africanus. Ann. Soc. Belge Med. Trop.*, V, 119.

GUGLIELMI, G. (1899). Un caso di malaria nel cavallo. *Clin. Vet.*, Milan, 220.

GUIART, J. (1903). Sur un nouvel infusoire parasite de l'homme. *C. R. Soc. Biol.*, LV, 245.

—— (1922). Coccidiose. Roger, Widal, & Teissier's *Nouveau Traité de Médecine*, V, 243.

GUILLEBEAU, A. (1893). Über das Vorkommen von *Coccidium oviforme* bei der rothen Ruhr des Rindes. *Mitt. Naturf. Ges. Bern*, 8; *Schweiz. Arch. Tierheilk.*, 1894, XXXVI, 169.

—— (1916). Parasitisches Vorkommen von *Eimeria stiedæ* in der Leber des Hundes. *Schweiz. Arch. Tierheilk.*, LVIII, 596.

GUNN, H. (1922). *Councilmania lafleuri* not a new amœba. *Journ. Parasit.*, IX, 24.

GURLEY, R. R. (1893). On the classification of Myxosporidia, a group of Protozoan parasites infesting fishes. *Bull. U.S. Fish. Comm.*, XI, 407.

—— (1894). The Myxosporidia or psorosperms of fishes, and the epidemics produced by them. *Rept. U.S. Fish. Comm.*, V, 65.

HADLEY, P. B. (1909). Studies in avian coccidiosis. I. White diarrhœa of chicks. II. Roup in fowls. *Centralbl. Bakt.*, I. Abt.. L, 348.

—— (1910). Studies in avian coccidiosis. III. Coccidiosis in the English sparrow and other wild birds. *Ibid.*, I. Abt., LVI, 522.

—— (1911). *Eimeria avium:* a morphological study. *Arch. Protist.*, XXIII, 7.

—— (1916). The rôle of the flagellated Protozoa in infective processes of the intestine and liver. *Rhode Isl. Agric. Exp. Stat.*, Bull. No. 166.

—— (1917). The case of Trichomonas. *Amer. Natural.*, LI, 209.

HADLEY, P. B., & AMISON, E. E. (1911). Further studies on blackhead in turkeys. *Centralbl. Bakt.*, I. Abt., LVIII, 34.

HADWEN, S. (1922). Cyst-forming Protozoa in reindeer and caribou, and a sarcosporidian of the seal (*Phoca richardi*). *Journ. Amer. Vct. Med. Assoc.*, LXI, 374.

HAECKEL, E. (1866). *Generelle Morphologie.* Berlin.

HAENDEL, L., & JOETTEN, K. W. (1920). Ueber chemotherapeutische Versuche mit " 205 Bayer " einem neuen trypanoziden Mittel von besonderer Wirkung. *Berlin. Klin. Woch.*, LVII, 821.

HAGENMÜLLER, P. (1898). Sur une nouvelle coccidie, parasite du *Gongylus ocellatus*. *C. R. Soc. Biol.*, L, 73.

—— (1898a). Sur une nouvelle coccidie diplosporée (*Diplospora laverani* Hgm.) parasite d'un ophidien. *Ibid.*, L, 309.

HAKE, T. G. (1839). *A treatise on varicose capillaries, as constituting the structure of carcinoma of the hepatic ducts, and developing the law and treatment of morbid growths. With an account of a new form of pus globule.* London.

HALBERSTÄDTER, L., & PROWAZEK, S. VON (1907). Untersuchungen über die Malariaparasiten der Affen. *Arb. K. Gesundheitsamte*, XXVI, 37.

HALL, M. C. (1917). Parasites of the dog in Michigan. *Journ. Amer. Vet. Med. Assoc.*, IV, 383.

HALL, M. C., & WIGDOR, M. (1918). Canine coccidiosis, with a note regarding other Protozoan parasites from the dog. *Ibid.*, LIII, 64.

HAMBURGER, C. (1912). Über einige parasitische Flagellaten. (Vorl. Mitt.) *Verhandl. Naturh.- Med. Ver. Heidelberg*, XI, 211.

HARRIS, H. F. (1901). Experimentell bei Hunden erzeugte Dysenterie. *Arch. Path. Anat.*, CLXVI, 67.

HARTMANN, M. (1907). Das System der Protozoen. *Arch. Protist.*, X, 139.

—— (1907a). [Note on *Amœba froschi* and *Amœba lacertæ*.] *Ibid.*, X, 314.

—— (1908). Eine neue Dysentericamöbe. *Entamœba tetragena* (Viereck) syn. *Entamœba africana* (Hartmann). *Arch. Schiffs.- u. Trop.-Hyg.* (Beih. 5), XII. 117.

—— (1909). Autogamie bei Protisten und ihre Bedeutung für das Befruchtungsproblem. *Arch. Protist.*, XIV, 264.

—— (1910). Notiz über eine weitere Art der Schizogonie bei *Schizotrypanum cruzi* (Chagas). *Ibid.*, XX, 361.

—— (1910a). Protozoologie. Kisskalt u. Hartmann's *Prakt. d. Bakt. u. Protozool.* Jena.

—— (1910b). Nova ameba intestinal, *Entamœba testudinis* n. sp., *Mem. Inst. O. Cruz*, II, 3.

—— (1913). Morphologie und Systematik der Amöben. Kolle u. Wassermann's *Handb. d. Path. Mikroorg.*, 2 edition, VII, 607.

—— (1914). Bemerkungen über *Amœba lacertœ* Hartmann, eine Antwort an Clifford Dobell. *Arch. Protist.*, XXXIV, 336.

—— (1917). Ueber die Schizogonie von *Schyzotrypanum cruzi. Ibid.*, XXXVIII, 113.

—— (1921). Rhinosporidium. Prowazek u. Nöller's *Handb. d. path. Prot.*, Leipzig, 1387.

HARTMANN, M., & CHAGAS, C. (1910). Vorläufige Mitteilung über Untersuchungen an Schlangen-hämogregarinen. *Arch. Protist.*, XX, 351.

—— —— (1910a). Flagellaten Studien. *Mem. Inst. O. Cruz*, II, 64.

—— —— (1910b). Über die Kernteilung von *Amœba hyalina* Dang. *Ibid.*, II. 159.

HARTMANN, M., & JOLLOS, V. (1910). Die Flagellatenordnung " Binucleata." Phylogenetische Entwicklung und systematische Einteilung der Blutprotozoen. *Arch. Protist.*, XIX, 81.

HARTMANN, M., & NÄGLER, K. (1908). Kopulation bei *Amœba diploidea* n. sp. mit Selbständig-bleiben der Gametenkerne während des ganzen Lebenszyklus. *Sitz. Ges. Nat. Freunde*, 112.

HARTMANN, M., & NÖLLER, W. (1918). Untersuchungen über die Cytologie von *Trypanosoma theileri. Arch. Protist.*, XXXVIII, 355.

HARTMANN, M., & PROWAZEK, S. VON (1907). Blepharoplast, Caryosom und Centrosom. Ein Beitrag zur Lehre von der Doppelkernigkeit der Zelle. *Ibid.*, X, 306.

HARTMANN, M., & SCHILLING, C. (1917). *Die pathogenen Protozoen und die durch sie verursachten Krankheiten.* Berlin.

HARTMANN, M., & SCHÜSSLER. H. (1913). Flagellata. *Handw. b. d. Naturwiss.* Jena, III, 1179.

HARTOG, M. (1906). The Protozoa. *Cambridge Nat. Hist..* I, 3.

HARZ (1886). In Koch's *Encyclopädie der gesamten Tierheilkunde*, IV, 89.

HASSALL, A. H. (1859). On the development and signification of *Vibrio lineola, Bodo urinarius*, and on certain fungoid and other organic productions generated in alkaline and albuminous urine. *Lancet*, II, 503.

HASSELMANN, G. (1918). Sobre os ciliados intestinaes dos mammiferos. *Brazil-Medico*, XXXII, 41, 81.

HAUGHWOUT, F. G. (1921). A case of human coccidiosis detected in the Philippine Islands, with remarks on the development and vitality of the cysts of *Isospora hominis* (Rivolta). *Philippine Journ. Sci.*, XVIII, 449.

—— (1921a). Coccidiosis in man as a possible sanitary problem in the United States. *Journ. Amer. Med. Assoc.*, LXXVII, 940.

HAUGHWOUT, F. G., & HORILLENO, F. S. (1920). The intestinal animal parasites found in one hundred sick Filipino children. *Philippine Journ. Sci.*, XVI, 1.

HAYASHI, N. (1920). Ætiology of tsutsugamushi disease. *Journ. Parasit.*, VII, 53.

HEARSEY, H. (1909). *Medical Report for the year 1908-09*. Nyasaland. *Bull. Sleep. Sickn. Bur.*, I, 205, 347.

HECKENROTH, F., & BLANCHARD, M. (1913). Transmission du *Trypanosoma gambiense* par des moustiques (*Mansonia uniformis*). *Bull. Soc. Path. Exot.*, VI, 442.

HEGLER, C., & PROWAZEK, S. VON (1913). Untersuchungen über Fleckfieber. Vorl. Ber. *Berlin. Klin. Woch.*, L, 2035.

HEGNER, R. W. (1920). The relation of medical zoology to public health problems. *Journ. Amer. Med. Assoc.*, LXXV, 1607.

—— (1921). Measurements of *Trypanosoma diemyctyli* from different hosts and their relation to specific identification, heredity, and environment. *Journ. Parasit.*, VII, 105.

——(1921a). *Cytamœba bacterifera* in the red blood-cells of the frog. *Ibid.*, VII, 157.

—— (1922). The systematic relationship of *Giardia lamblia* Stiles, 1915, from man and *Giardia agilis* Kunstler, 1882, from the tadpole. *Amer. Journ. Hyg.*, II. 435.

—— (1922a). A comparative study of the giardias living in man, rabbit, and dog. *Ibid.*, II, 442.

—— (1923). Observations on a *Balantidium* from a Brazilian monkey, *Cebus variegatus*, E. Geoffr., with special reference to chromosome-like bodies in the macronuclei. *Ibid.*, III, 252,

—— (1923a). Giardias from wild rats and mice and *Giardia caviœ* n. sp. from the guinea-pig. *Ibid.*, III, 345.

—— (1923b). Nuclear division within the cysts of the human intestinal Protozoon, *Chilomastix mesnili*. *Ibid.*, III, 349.

—— (1923c). Observations and experiments on Euglenoidina in the digestive tract of frog and toad tadpoles. *Biol. Bull. Marine Biol. Lab. Woods Hole, Mass.*, XLV, 162.

—— (1924). *Giardia* and *Chilomastix* from monkeys, *Giardia* from the wild cat, and *Balantidium* from the sheep. *Journ. Parasit.*, XI, 75.

—— (1925). *Trichomonas vaginalis* Donné. *Amer. Journ. Hyg.*, V, 302.

—— (1925a). *Giardia felis*, n. sp., from the domestic cat and giardias from birds. *Ibid.*, V, 258.

—— (1926). *Endolimax cavae* n. sp. from the guinea-pig and *Endolimax januae* n. sp. from the domestic fowl. *Journ. Parasit.*, XII., 146.

HEGNER, R. W., & BECKER, E. R. (1922). The diagnosis of intestinal flagellates by culture methods. *Ibid.*, IX, 15.

HEGNER, R. W., & PAYNE, G. C. (1921). Surveys of the intestinal Protozoa of man in health and disease. *Scient. Monthly*, January, 47.

HEGNER, R. W., & TALIAFERRO, W. H. (1924). *Human protozoology*. New York.

HEGNER, R. W., & WU, H. F. (1921). An analysis of the relation between growth and nuclear division in a parasitic infusorian, *Opalina* sp. *Amer. Natural.*, LV, 335.

HEHEWERTH, F. H., & KOP, W. A. (1921). The Wassermann test in patients affected with malaria in the tropics. *Journ. Hyg.*, XIX, 277.

HELM (1924). Beitrag zum Anaplasmen-Problem. *Zeitschr. Infektionskr. d. Haust.*, XXV, 199.

HENLE (1835). Über die Gattung *Brachiobdella* und über die Bedeutung der inneren Geschlechtstheile bei den Anneliden und hermaphroditischen Schnecken. *Arch. Anat. & Phys.*, II, 574.

HENLE, J. (1845). Ueber die Gattung *Gregarina*. *Ibid.*, XIII, 369.

HENNEGUY, L. F. (1883). Sur un infusoire flagellé, ectoparasite des poissons. *C. R. Acad. Sci.*, XCVI, 658.

—— (1884). Note sur un infusoire flagellé ectoparasite de la truite. *Arch. Zool. Exp.*, II, 403.

HENRY, A., & LEBLOIS, C. (1925). Essai de classification des coccidies disporocystées tétrazoïques. *C. R. Acad. Sci.*, CLXXX, 700.

—— (1926). Essai de classification des coccidies de la famille des Diplosporidae Léger, 1911. *Ann. Parasit. Hum. & Comp.*, IV, 22.

HENRY, H. (1910). On the Hæmoprotozoa of British sea-fish. *Journ. Path. & Bact.*, XIV, 463.

—— (1913). A new hæmosporidian parasite from *Scomber scomber*, the common mackerel. *Ibid.*, XVIII, 228.

—— (1913a). A hæmogregarine and a leucocytozoon from *Gadus œglefinus*. *Ibid.*, XVIII, 232.

—— (1913b). An intracorpuscular parasite in the blood of *Cottus bubalis* and *Cottus scorpius*. *Ibid.*, XVIII, 224.

—— (1917). The "hæmogregarine" of trench fever. *Brit. Med. Journ.*, II, 739.

HERTIG, M., & WOLBACH, S. B. (1924). Studies on Rickettsia-like micro-organisms in insects. *Journ. Med. Res.*, XLIV, 329.

HERTWIG, R. (1876). Ueber *Podophrya gemmipara*, nebst Bemerkungen zum Bau und zur systematischen Stellung der Acineten. *Morphol. Jahrb.*, I, 20.

—— (1889). Ueber die Conjugation der Infusorien. *Abh. Bayer. Akad. Wiss.*, XVII, 151.

—— (1898). Ueber Kerntheilung, Richtungskörperbildung und Befruchtung von *Actinosphæ-rium eichhorni*. *Ibid.*, XIX, 631.

—— (1899). Ueber Encystierung und Kernvermehrung bei *Arcella vulgaris*. *Kupffer's Festschr.*

HESSE, E. (1892). Die rothe Ruhr des Rindes. *Schweiz. Arch. Tierheilk.*, XXXIV, 105.

—— (1904). *Thelohania legeri* n. sp., microsporidie nouvelle, parasite des larves d'*Anopheles maculipennis* Meig. *C. R. Soc. Biol.*, LVII, 570.

—— (1904a). Sur le développement de *Thelohania legeri* Hesse. *Ibid.*, LVII, 571.

—— (1905). Sur *Myxocystis mrazeki* Hesse, microsporidie de *Limnodrilus hoffmeisteri*. *Ibid.*, LVIII, 12.

—— (1910). *Trypanoplasma vaginalis* n. sp., parasite du vagin de la sangsue. *C. R. Acad. Sci.*, CLI, 504.

—— (1911). Sur le genre *Adelea* à propos d'une nouvelle coccidie des oligochètes. *Arch. Zool. Exp.*, VII (N. & R.), xv.

—— (1918). *Caulleryella anophelis* n. sp. schizogrégarine parasite des larves d'*Anopheles bifurcatus* L. *C. R. Acad. Sci.*, CLXVI, 569.

HICKSON, S. J. (1903). The Infusoria or Corticata heterokaryota. Lankester's *Treatise on Zoology*, Part I, 361.

HILL, J. (1752). *History of animals*. London.

HINDLE, E. (1909). The life-history of *Trypanosoma dimorphon*, Dutton & Todd. *Univ. California Publ. Zool.*, VI, 127.

—— (1921). Notes on Rickettsia. *Parasitology*, XIII, 152.

HINES, L. E. (1923). *Endamœba histolytica* in seminal fluid in a case of amœbic dysentery. *Journ. Amer. Med. Assoc.*, LXXXI, 274.

HINKELMANN, A. J. (1919). Pathogenicity, cultivation and reproduction of Protozoon parasites. *New York Med. Journ.*, CIX, 255.

HINTZE, R. (1901). Lebensweise und Entwicklung von *Lankesterella minima* Chaussat. *Zool. Jahrb.*, *Abt. f. Anat. u. Ontog.*, XV, 693.

HLAVA (1887). Předbežné sdĕleni. [On dysentery; in Bohemian.] *Zeitschr. böhm. Aerzte in Prag.* [See " Uplavici, O.," by Kartulis. *Centralbl. Bakt.*, 1887, I. Abt., I, 537.]

HOARE, C. A. (1918). *Hœmogregarina tigrinæ* n. sp. from *Natrix tigrina* Boie. *Arch. Russes Anat. Histol., Embryol.*, II, 129.

—— (1921). On some new hæmogregarines from British East Africa. *Parasitology*, XII, 315. (Part IV. published January, 1921.)

—— (1921a). Some observations and experiments on insect flagellates, with special reference to artificial infection of vertebrates. *Ibid.*, XIII, 67.

—— (1922). Trypanosomiasis in British sheep. (Preliminary communication.) *Trans. Roy. Soc. Trop. Med. & Hyg.*, XVI, 188.

—— (1923). An experimental study of the sheep trypanosome (*T. melophagium* Flu, 1908) and its transmission by the sheep ked (*Melophagus ovinus* L.). *Parasitology*, XV, 365.

—— (1924). A note on the specific name of the herpetomonad of the house fly. *Trans. Roy. Soc. Trop. Med. & Hyg.*, XVII, 403.

—— (1924a). *Hepatozoon adiei*, n. sp., a blood parasite of an Indian eagle. *Ibid.*, XVIII, 63.

—— (1926). [Infection of kitten by injection of cysts of *Entamœba histolytica per rectum*.] *Ibid.*, XIX, 277.

HOBMAIER, M. (1922). Globidiuminfektion beim Fohlen. *Berlin. Tierärztl. Woch.*, 100.

HÖFFLE (1850). *Chem. u. Mikrosk. am Krankenbett.*, 61.

HOFER, B. (1904). Die Schlafsucht des Karpfens. *Allg. Fisch.-Zeit.*, XIX, 48.

HOFFMANN, W. H. (1922). Zur Vererbung von Krankheitserregern in den übertragenden Insekten nach Beobachtungen an *Rhodnius prolixus*. *Münch. Med. Woch.*, LXIX, 1623.

HOGUE. M. J. (1914). Studies in the life-history of an amœba of the limax group—*Vahlkampfia calkensi*. *Arch. Protist.*, XXXV, 154.

—— (1921). Studies on the life-history of *Vahlkampfia patuxent* n. sp., parasitic in the oyster, with experiments regarding its pathogenicity. *Amer. Journ. Hyg.*, I, 321.

—— (1921a). The cultivation of *Trichomonas hominis*. *Amer. Journ. Trop. Med.*, I, 211.

—— (1921b). *Waskia intestinalis*: its cultivation and cyst formation. *Journ. Amer. Med. Assoc.*, LXXVII, 112.

—— (1922). A study of *Trichomonas hominis*: its cultivation, its inoculation into animals, and its staining reaction to vital dyes. *Johns Hopkins Hosp. Bull.*, XXXIII, 437.

HOLLANDE, CH. (1912). Sur l' " *Herpetomonas emphyti* " n. sp., parasite d'une larve d'hymenoptère, l' "*Emphytus cinctus* " Klüg. *C. R. Soc. Biol.*, LXXII, 940.

HOLMES, F. O. (1923). Observations on the cysts of *Endamœba cobayœ*. *Journ. Parasit.*, X, 47.

HOLMES, J. D. E. (1910). The treatment of surra in horses by means of arsenic and its derivatives. Thirty-two cases of successful treatment. *Journ. Trop. Vet. Sci.*, V, 1.

HORNBY, H. E. (1919). Some notes on the use of tartar emetic in the treatment of domestic animals affected with trypanosomiasis. *Vet. Journ.*, LXXV, 89.

—— (1921). Trypanosomes and trypanosomiases of cattle. *Journ. Comp. Path. & Therap.*, XXXIV, 211.

—— (1919a). The trypanosomes found in the domestic mammals in South-Central Africa *Vet. Journ.*, LXXV, 128.

HOVASSE, R. (1924). *Trypanoplasma sagittæ*, sp. nov. *C. R. Soc. Biol.*, XCI, 1254.

HOWITT, B. F. (1925). The cultivation of *Endamœba gingivalis* (Gros). *Univ. California Publ. Zool.*, XXVIII, 65.

HUBER (1903). [Demonstration,"Dysenterieamöben,"in Ver. f. innere Med., Berlin.] *Deutsch. Med. Woch.*, XXIX, 267.

—— (1909). Untersuchungen über Amöbendysenterie. *Zeitschr. Klin. Med.*, LXVII, 262.

HUNTEMÜLLER (1914). Neuartige Parasitenbefunde bei der Jerichobeule. *Centralbl. Bakt.*, I. Abt., LXXIII, 137.

HUTCHEON, D. (1893). Malignant jaundice or bilious fever of the dog. *Agric. Journ.*, Dept. Agric. Cape Colony, VI, 476.

HUTCHEON, D., & ROBERTSON, W. (1902). Malarial catarrhal fever of sheep. *Vet. Record*, 629.

IKEDA, I. (1912). Studies on some sporozoon parasites of Sipunculoids. I. The life-history of a new Actinomyxidian, *Tetractinomyxon intermedium* g. et sp. nov. *Arch. Parasit.*, XXV, 240.

—— (1914). Studies on some sporozoan parasites of Sipunculoids. II. *Dobellia binucleata* n. g., n. sp., a new coccidian from the gut of *Petalostoma minutum* Keferstein. *Ibid.*, XXXIII. 205.

ILOWAISKY, S. (1922). Amöbenhepatitis bei *Rana temporaria. Arch. Soc. Russe Protist.*, Moscow, I, 82.

INCHIOSTRI, H. (1912). Vorkommen und Formen der " *Piroplasmosis ovis* " in Dalmatien. *Öster. Woch. Tierheilk.*, XXXVII. 289.

ITURBE, J. (1918). Presencia del *Leptomonas davidi* (Lafont) en la savia de la *Euphorbia hypericifolia. Gaceta Med. Caracas*, XXV, 173.

ITURBE, J., & GONZALEZ, E. (1916). *El paludismo de las aves en Venezuela.* Caracas.

—— —— (1921). *Plasmodium gonzalezi* nov. sp. Huésped: *Anolis biporcatus* Bocourt. *Gaceta Med. Caracas.* XXVIII, 275.

IWANOW, E. (1925). Le sort du *Trypanosoma equiperdum* dans la cavité générale de *Galleria mellonella. C. R. Soc. Biol.*, XCII, 345.

JACOBY, M. (1909). Ueber die Herstellung von serumfesten Trypanosomenstämmen. *Med. Klin.*, V, 252.

—— (1909a). Ueber Serumfestigkeit und die Einwirkung von menschlichem Blutserum auf Trypanosomen. *Zeitschr. Imm.-Forsch.*, II, 689.

JACQUEMET, M. (1903). Sur la systématique des coccidies des céphalopodes. *Arch. Protist.*, II, 190.

JAKOBY, M., & SCHAUDINN, F. (1899). Ueber zwei neue Infusorien im Darm des Menschen. *Centralbl. Bakt.*, I. Abt., XXV, 487.

JAMES, S. P. (1903). A report of the anti-malarial operations at Mian Mir (1901-02). *Repts. Malar. Cttee. Roy. Soc.*, VIII, 27.

—— (1905). On a parasite found in the white blood corpuscles of dogs. *Sci. Mem. Off. Med. & San. Dep. Gov. India*, No. 14.

—— (1905a). A new leucocytozoon of dogs. *Brit. Med. Journ.*, I, 1361.

JAMES, S. P. (1926). [Malaria in mosquitoes.] *Trans. Roy. Soc. Trop. Med. & Hyg.*, XIX, 278.

JAMESON, A. P. (1914). A new phytoflagellate (*Parapolytoma satura* n. g., n. sp.) and its method of nuclear division. *Arch. Protist.*, XXXIII, 21.

—— (1925). A new ciliate, *Charon ventriculi* n. g., n. sp., from the stomach of ruminants. *Parasitology*, XVII, 403.

JANCSÓ, N. (1904). Zur Frage der Infektion des *Anopheles claviger* mit Malariaparasiten bei niederer Temperatur. *Centralbl. Bakt.*, I. Abt., XXXVI, 624.

—— (1921). Experimentelle Untersuchungen ueber die die Malariainfektion des Anopheles und des Menschen beeinflussenden Umstände. *Arch. Schiffs.- u. Trop.-Hyg.*, XXV (Beih. 2), 5.

JANICKI, C. (1908). Contribuzione alla conoscenza di alcuni protozoi parassiti della *Periplaneta orientalis* (*Lophomonas blattarum* Stein, *L. striata* Bütschli, *Amœba blattæ* Bütschli). *Atti R. Accad. Lincei, Rendic.*, XVII, ii, 140.

—— (1909). Ueber Kern und Kernteilung bei *Entamœba blattæ* Bütschli. *Biol. Centralbl.*, XXIX, 381.

—— (1911). Zur Kenntnis des Parabasalapparates bei parasitischen Flagellaten. *Ibid.*, XXXI, 321.

—— (1912). Paramöbenstudien (*P. pigmentifera* Grassi und *P. chætognathi* Grassi). *Zeitschr. Wiss. Zool.*, CIII, 449.

—— (1915). Untersuchungen an parasitischen Flagellaten. II. Teil. Die Gattungen *Devescovina, Parajœnia, Stephenonympha, Calonympha.* Über den Parabasalapparat. Über Kernkonstitution und Kernteilung. *Ibid.*, CXII, 573.

JANOWSKI, W. (1897). Ueber Flagellaten in den menschlichem Fäces und über ihre Bedeutung für die Pathologie des Darmcanals. *Zeitschr. Klin. Med.*, XXXI, 442.

JEMMA, R. (1910). Sulla infezione spontanea da parasiti di Leishmann nei cani. *Pathologica*, II, 250.

—— (1912). Sulla Leishmaniosi del cane nei dintorni di Palermo. *Ibid.*, IV, 466.

JEMMA, R., DI CRISTINA, G., & CANNATA, S. (1910). Infezione sperimentale da *Leishmania infantum* nei cani. *Pediatria*, VIII, 241.

JENNINGS, H. S. (1916). Heredity, variation and the results of selection in the uniparental reproduction of *Difflugia corona*. *Genetics*, I, 407.

—— (1920). *Life and death, heredity and evolution in unicellular organisms*. Boston.

JEPPS, M. W. (1921). Notes on the intestinal Protozoa of 971 men at the University War Hospital, Southampton. *Journ. R. Army Med. Corps*, XXXVII, 366.

—— (1923). Observations on human intestinal Protozoa in Malaya. *Parasitology*, XV, 213.

—— (1923a). Protozoa of the human mouth in Malaya, *Entamœba gingivalis* and *Trichomonas* sp. *Ibid.*, XV, 343.

JEPPS, M., & DOBELL, C. (1918). *Dientamœba fragilis*, n. g., n. sp., a new intestinal amœba from man. *Ibid.*, X, 352.

JOB, E., & DUMAS (1919). Etude sur les états dysentériques de l'armée d'Orient. *Arch. Med. & Pharm. Milit.*, LXXI, 209.

JOB, E., & HIRTZMANN, L. (1918). L'évolution d'*Amœba dysenteriæ* et l'histopathogénie des abcès du foie. *Bull. & Mém. Soc. Méd. Hôpit. Paris*, XLII, 928.

—— —— —— (1919). Paludisme et infections typhoïdes. *Ibid.*, XLIII, 581.

—— —— (1919a). Paludisme et diarrhée. *Ibid.*, XLIII, 241.

—— —— —— (1919b). Dysenterie bacillaire et paludisme. *Ibid.*, XLIII, 714.

JOHNE (1882). Bericht über das Veterinärwesen im Königreich Sachsen, p. 66.

JOHNSON (1903). Preliminary note on ictero-hæmaturia of sheep in West Montana. *Proc. Amer. Vet. Med. Assoc.*, 301.

JOHNSON, E. A. (1907). A trypanosome found in the River Murray turtle (*Chelodina longicollis*). *Australas. Med. Gaz.*, XXVI, 26.

JOHNSON, W. B. (1920). *Annual medical and sanitary report of the Northern Provinces, Nigeria, for the year ending December 31, 1918*.

JOHNSTON, J. E. L. (1917). On some peculiar bodies found in the blood of certain African snakes. *Journ. Trop. Med. & Hyg.*, XX, 241.

JOHNSTON, T. H. (1909). On some hæmogregarines from Australian reptiles. *Proc. Lin. Soc. N. S. Wales*, XXXIV, 400.

—— (1910). Hæmogregarinidæ. *Rept. Govt. Bur. Microbiol., 1909*, Sydney, 41.

JOHNSTON, T. H., & CLELAND, J. B. (1909). On a new melanin-producing hæmatozoon from an Australian tortoise. *Journ. & Proc. Roy. Soc. N. S. Wales*, XLIII, 97.

—— —— —— (1909a). Notes on some parasitic Protozoa. *Ibid.*, XXXIV, 501.

—— —— —— (1910). The hæmatozoa of Australian reptilia. No 1. *Ibid.*, XXXV, 677.

—— —— —— (1912). The hæmatozoa of Australian reptilia. No. 2. *Ibid.*, XXXVI, 479. (Part III, though bound in 1911 vol., was issued in 1912.)

JOLLOS, V. (1909). Multiple Teilung und Reduktion bei *Adelea ovata* (A. Schneider). *Arch. Protist.*, XV, 249.

—— (1910). Bau und Vermehrung von *Trypanoplasma helicis*. *Ibid.*, XXI, 103.

—— (1911). Studien über parasitische Flagellaten. I. *Monocercomonas cetoniæ*, n. sp. *Ibid.* XXIII, 311.

—— (1916). Neuere Untersuchungen über die Darmamöben des Menschen. *Ibid.*, XXXVI, 364.

—— (1917). Untersuchungen zur Morphologie der Amöbenteilung. *Ibid.*, XXXVII, 229.

JOLYET, F., & NABIAS, B. DE (1891). Sur un hématozoaire du lapin domestique. *Journ. Méd. Bordeaux*, XX, 325.

JONES, H. (1846). Examen microscopique d'un foie de lapin altéré. *Arch. Gén. Méd. Journ. Compl. Sci. Med.* XI. (*Arch. Anat. Gén. & Phys.*, 18.)

JOUKOFF, N. M. (1913). Culture du parasite de la malaria. *C. R. Soc. Biol.*, LXXIV, 136.

JOWETT, W. (1911). Coccidiosis of the fowl and calf. *Journ. Comp. Path. & Therap.*, XXIV, 207.

—— (1911a). Blackhead, infectious entero-hepatitis or typhlo-hepatitis, a disease of young turkeys. *Ibid.*, XXIV, 289.

JOYEUX, C. (1913). Note sur quelques protozoaires sanguicoles et intestinaux observés en Guinée française. *Bull. Soc. Path. Exot.*, VI, 612.

KARTULIS, [S.] (1885). Ueber Riesen-Amöben (?) bei chronischer Darmentzündung der Ägypter. *Arch. Path. Anat.*, XCIX, 145.

—— —— (1886). Zur Aetiologie der Dysenterie in Aegypten. *Ibid.*, CV, 521.

—— —— (1891). Einiges über die Pathogenese der Dysenterieamöben. *Centralbl. Bakt.*, I. Abt., IX, 365.

KARTULIS, [S.] (1893). Ueber pathogene Protozoen bei dem Menschen. *Zeitschr. Hyg. u. Infekt.*, XIII, 1.

—— (1906). Die Amöbendysenterie. Kolle & Wassermann's *Handb. d. path. Mikroorg.*, 1st edition, Ergänzungsband I, 347.

—— (1913). Die Amöbendysenterie. *Ibid.*, 2nd edition, VII, 651.

KASPAREK, T. (1895). Beitrage zur den Infektionsversuchen mit Sarcosporidien. *Centralbl. Bakt.*, I. Abt., XVIII, 327.

KAUFFMANN, W. (1847). Analecta ad tuberculorum et entozoorum cognitionem. (Diss. Inaug.) Berol.

KEFERSTEIN, W. (1861). Über parasitische Pilze aus *Ascaris mystax*. *Zeitschr. Wiss. Zool.*, XI, 135.

KEFERSTEIN, W., & EHLERS, E. (1860). Beiträge zur Kenntniss der Geschlechtsverhältnisse von *Helix pomatia*. *Ibid.*, X, 253.

KEILIN, D. (1914). Une nouvelle schizogrégarine *Caulleryella aphiochœtœ*, n. g., n. sp., parasite intestinal d'une larve d'un diptère cyclorhaphe (*Aphiochœta rufipes* Meig.). *C. R. Soc. Biol.*, LXXVI, 768.

—— (1917). Une nouvelle entamibe, *Entamœba mesnili*, n. sp., parasite intestinal d'une larve d'un diptère. *Ibid.*, LXXX, 133.

—— (1921). On the life-history of *Helicosporidium parasiticum*, n. g., n. sp., a new type of protist parasitic in the larva of *Dasyhelea obscura* Winn. (Diptera, Ceratopogonidæ) and in some other arthropods. *Parasitology*, XIII, 97.

—— (1921a). On a new ciliate, *Lambornella stegomyiœ*, n. g., n. sp., parasitic in the body cavity of the larvæ of *Stegomyia scutellaris* Walker (Diptera, Nematocera, Culicidæ). *Ibid.*, XIII, 216.

—— (1923). On a new schizogregarine, *Schizocystis legeri*, n. sp., an intestinal parasite of dipterous larvæ (*Systenus*). *Ibid.*, XV, 103.

—— (1923a). The structure and life-history of *Lipotropha*, n. g., a new type of schizogregarine, parasitic in the fat body of a dipterous larva (*Systenus*). *Proc. Cambridge Philos. Soc.* (Biol. Sci.), I, 18.

KELLESBERGER, E. R. (1925). Note on a case of sleeping sickness in a child three weeks old. *Trans. Roy. Soc. Trop. Med. & Hyg.*, XIX, 81.

KELLICOTT, D. S. (1888). Observations on fresh-water infusoria. *Proc. Amer. Soc. Micr.*, X, 97.

KENT, W. S. (1880–1882). *A manual of the Infusoria*. London.

KÉRANDEL, J. (1909). Sur quelques hématozoaires observés au Congo (Haut-Sangha-Logone). *Bull. Soc. Path. Exot.*, II, 204.

KERZELLI, S. (1909). Piroplasmosis tarandi rangiferis. *Arch. Vet. Nauk*, No. 5.

KESSEL, J. F. (1923). Methods of obtaining amœba-free rats for experimental infection with intestinal amœbæ. *Univ. California Publ. Zool.*, XX, 401.

—— (1923a). Experimental infection of rats and mice with the common intestinal amœbæ of man. *Ibid.*, XX, 409.

—— (1924). The distinguishing characteristics of the parasitic amœbæ of culture rats and mice. *Ibid.*, XX, 489.

—— (1924a). The experimental transfer of certain intestinal Protozoa from man to monkeys. *Proc. Soc. Exp. Biol. & Med.*, XXII, 206.

—— (1925). The ingestion of erythrocytes by *Trichomonas hominis* and its occurrence in the pus of an amœbic liver abscess. *Journ. Parasit.*, XI, 151.

KEYSSELITZ, G. (1904). Über *Trypanophis grobbeni* (*Trypanosoma grobbeni* Poche). *Arch. Protist.*, III, 367.

—— (1906). Generations- und Wirtwechsel von *Trypanosoma borelli* Laveran et Mesnil. *Ibid*, VII, 1.

—— (1908). Die Entwicklung von *Myxobolus pfeifferi* Th. Teile 1-2. *Ibid.*, XI, 252.

KING, A. F. A. (1883). Insects and disease—mosquitoes and malaria. *Pop. Sci. Monthly*, New York, September.

KING, S. D., & GATENBY, J. B. (1926). Note on certain new bodies in *Opalina ranarum*, presumed to represent the Golgi elements. *Quart. Jl. Micro. Sci.*, LXX, 217.

KING, W. V. (1906). Experimental transmission of Rocky Mountain spotted fever by means of the tick. Preliminary note. *U. S. Pub. Health Rep.*, XXI, 863.

—— (1917). The effect of cold on the malaria parasites in the mosquito host. *Journ. Exp. Med.*, XXV, 495.

KINGHORN, A., & YORKE, W. (1912). On the transmission of human trypanosomes by *Glossina morsitans* Westw., and on the occurrence of human trypanosomes in game. *Ann. Trop. Med. & Parasit.*, VI, 1, 269.

—— —— (1912a). Trypanosomes infecting game and domestic stock in the Luangwa valley, North-Eastern Rhodesia. *Ibid.*, VI, 301, 483.

—— —— (1912b). Trypanosomes obtained by feeding wild *Glossina morsitans* on monkeys in Luangwa valley, Northern Rhodesia. *Ibid.*, VI, 317.

—— —— (1912c). Further observations on the trypanosomes of game and domestic stock in North-Eastern Rhodesia. *Ibid.*, VI, 483.

KINGHORN, A., YORKE, W., & LLOYD, L. (1913). Final report of the Luangwa Sleeping Sickness Commission of the British South Africa Company, 1911-12. *Ibid.*, VII, 183.

KINOSHITA, K. (1906). Ueber die Verbreitung der Anophelen auf Formosa und deren Beziehungen zu den Malariakrankheiten. *Arch. Schiffs-u. Trop.- Hyg.*, X, 621, 676, 708, 741.

KISSKALT, K. (1905). Blutparasiten bei Fledermäusen. *Centralbl. Bakt.*, I. Abt., XL, 213.

KITASHIMA, T., & MIYAJIMA, M. (1918). Studien über die tsutsugamushikrankheit. *Kitasato Arch.*, II, 91, 237.

KLEBS, G. (1892). Flagellatenstudien—I & II. *Zeitschr. Wiss. Zool.*, LV, 265, 353.

KLEINE, F. K. (1909). Positive Infektionsversuche mit *Trypanosoma brucei* durch *Glossina palpalis*. *Deutsch. Med. Woch.*, XXXV, 469.

——(1909a). Weitere wissenschaftliche Beobachtungen über die Entwicklung von Trypanosomen in Glossinen. *Ibid.*, XXXV, 924.

——(1909b). Weitere Untersuchungen über die Aetiologie der Schlafkrankheit. *Ibid.*, XXXV, 1257.

——(1909c). Weitere Beobachtungen uber Tsetsefliegen und Trypanosomen. *Ibid.*, XXXV, 1956.

——(1910). Trypanosomenbefunde am Tanganyika und andere Beobachtungen. *Ibid.*, XXXVI, 1400.

——(1914). Zur angeblichen Identität des *Tr. brucei* und *Tr. rhodesiense*. *Zeitschr. Hyg. u. Infekt.*, LXXVII, 184.

——(1919). Ueber die Ergebnisse der deutschen Schlafkrankheitsforschung. *Deutsch. Med. Woch.*, XLV, 729.

——(1919a). Beitrag zur Kenntnis des *Trypanosoma melophagium* Flu. *Deutsch. Tierarztl. Woch.*, XXVII, 408.

——-(1923). Zur Epidemiologie der Schlafkrankheit. *Deutsch. Med. Woch.*, XLIX, 505.

——(1924). On a recent expedition to Africa to investigate the action of " Bayer 205 " in trypanosomiasis. *Trans. Roy. Soc. Trop. Med. & Hyg.*, XVII, 445.

KLEINE, F. K., & ECKARD, B. (1913). Ueber die Bedeutung der Haustiere und des Wildes für die Verbreitung der Schlafkrankheit. *Zeitschr. Hyg. u. Infekt.*, LXXV, 118.

KLEINE, F. K., & FISCHER, W. (1911). Die Rolle der Säugetiere bei der Verbreitung der Schlafkrankheit und Trypanosomenbefunde bei Säugetieren am Tanganyka. *Ibid.*, LXX, 1.

————(1912). Schlafkrankheit und Tsetsefliegen. *Ibid.*, LXXIII, 253.

————(1922). Bericht über die Prüfung von " Bayer 205 " in Afrika. *Deutsch. Med. Woch.*, XLVIII, 1693.

————(1923). 2. Bericht über Prüfung von " Bayer 205 " in Afrika. *Ibid.*, XLIX, 1039.

KLEINE, F. K., & TAUTE, M. (1911). Ergänzungen zu unseren Trypanosomenstudien. *Arb. K. Gesundheitsamte*, XXXI, 321.

KLIGLER, I. J. (1924). On the cultivation and biological characters of *Leishmania tropica*. *Amer. Journ. Trop. Med.*, IV, 69.

——(1926). The cultural and serological relationship of *Leishmania*. *Trans. Roy. Soc. Trop. Med. & Hyg.*, XIX, 330.

KLIPPEL & MONIER-VINARD (1922). Premier cas de kala-azar d'origine marocaine. Guérison par l'acétyl-amino-phényl stibinate de soude (stibényl). *Bull. & Mém. Soc. Méd. Hôpit. Paris*, XLVI, 76.

KLOSS, H. (1855). Ueber Parasiten in der Niere von *Helix*. *Abh. Senckenb. Naturf. Ges.*, I, 189.

KNIGHTON, J. E. (1922). *Lamblia intestinalis*, with report of cases. *Southern Med. Journ.*, XV, 457.

KNOWLES, R. (1919). Notes on a monkey plasmodium and on some experiments in malaria. *Indian Journ. Med. Res.*, VII, 195.

——(1920). A study of kala-azar. *Ibid.*, VIII. 140, 162.

——(1923). *Plasmodium ovale*. *Indian Med. Gaz.*, LVIII, 276.

——(1923a). *Plasmodium tenue* and *P. ovale*. *Ibid.*, LVIII, 499.

——(1924). Report of Professor of Protozoology. *Ann. Rept. Calcutta Sch. Trop. Med.*, 1923, 64.

KNOWLES, R., & DAS GUPTA, B. M. (1924). A note upon a flagellate Protozoon found in the saliva. *Indian Journ. Med. Res.*, XI, 737.

————(1924a). The diagnosis of kala-azar by examination of thick blood-films. *Indian Med. Gaz.*, LIX, 438.

KNOWLES, R., NAPIER, L. E., & DAS GUPTA, B. M. (1923). The kala-azar transmission problem. *Ibid.*, LVIII, 321.

KNOWLES, R., NAPIER, L. E., & SMITH, R. O. A. (1924). On a herpetomonas found in the gut of the sand fly, *Phlebotomus argentipes*, fed on kala-azar patients. A preliminary note. *Ibid.*, LIX, 593.

KNUTH, P. (1909). Über die Morphologie des *Trypanosoma Frank*. *Zeitschr. Infektionskr. d. Haust.*, XVI, 39.

——(1909a). Eine *Herpetomonas* beim Reh. *Ibid.*, VI, 357.

——(1915). Über die Ätiologie der inneren Verblutung (Milzruptur) bei Rindern und über dei künstliche Züchtung von *Haemaphysalis cinnabarina*, dem wahrscheinlichen Überträger des Erregers dieser Krankheit. *Arch. Schiffs.- u. Trop.-Hyg.*, XIX, 185.

KNUTH, P. (1915a). Über Piroplasmen bei europäischen Rinder mit besonderer Berücksichtigung ihrer Ätiologie. *Ibid.*, XIX, 245.

—— (1922). Demonstration über in Deutschland gefundene Leukozytozoen der Hausgans. *Ibid.*, XXVI, 315.

KNUTH, P., & MAGDEBURG, F. (1922). Ueber ein durch Leukocytozoen verursachtes Sterben junger Gänse. *Berlin. Tierärztl. Woch.*, XXXVIII, 359.

KNUTH, [P.], & RICHTERS, [E.] (1913). Über die Vermehrung von *Piroplasma canis in vitro*. *Ibid.*, XXIX, 211.

KNUTH, P., & DU TOIT, P. J. (1921). Tropen-Krankheiten der Haustiere. Mense's *Handb. d. Trop.-Krankh.*. 2nd edition., VI.

KOCH, R. (1897). Über die Viehseuchen in Deutsch-Ostafrika. *Deutsch. Kolon.-Bl.*, No. 24, 719.

—— (1898). Berichte über die Ergebnisse der Expedition des Geheimer-medicinalrathes Dr. Koch im Schutzgebiete von Deutsch-Ostafrika. (Reprinted in *Centralbl. Bakt.*. I. Abt., XXIV, 200.)

—— (1898a). *Reiseberichte über Rinderpest, Bubonenpest in Indien und Afrika, Tsetse- oder Surrakrankheit, Texasfieber, tropische Malaria, Schwarzwasserfieber.* Berlin.

—— (1899). Ergebnisse der wissenschaftlichen Expedition des Geheimen Medicinalraths Professor Dr. Koch nach Italien zur Erforschung der Malaria. *Deutsch. Med. Woch.*, XXV, 69.

—— (1899a). Über die Entwicklung der Malariaparasiten. *Zeitschr. Hyg. u. Infekt.*, XXXII, 1.

—— (1900). II. Bericht ueber die Tätigkeit der Malariaexpedition. *Deutsch. Med. Woch.*, XXVI, 88.

—— (1900a). Zusammenfassende Darstellung der Ergebnisse der Malariaexpedition. *Ibid.*, XXVI, 781, 801.

—— (1903). On Rhodesian red-water or African coast fever. *Journ. Comp. Path. & Therap.*, XVI, 273.

—— (1905). Vorlaufige Mitteilungen über die Ergebnisse einer Forschungsreise nach Ostafrika. *Deutsch. Med. Woch.*, XXXI, 1865.

—— (1906). Ueber den bisherigen Verlauf der deutschen Expedition zur Erforschung der Schlafkrankheit in Ostafrika. *Ibid.*, XXXII, Append. to No. 51.

—— (1906a). Beitrage zur Entwicklungsgeschichte der Piroplasmen. *Zeitschr. Hyg.- u. Infekt.*, LIV, 1.

—— (1907) Bericht über die Tätigkeit der deutschen Expedition zur Erforschung der Schlafkrankheit bis zum Nov. 25, 1906. *Deutsch. Med. Woch.*, XXXIII, 49.

—— (1907a). III. Bericht des Herrn Geh. Med.-Rat Prof. Robert Koch von der deutschen Expedition zur Erforschung der Schlafkrankheit. *Ibid.*, XXXIII, 1462.

—— (1907b). Schlussbericht uber die Tatigkeit der deutschen Expedition zur Erforschung der Schlafkrankheit. *Ibid.*, XXXIII, 1889.

KOCH, R., BECK, M., & KLEINE, F. (1909). *Bericht uber die Tätigkeit der zur Erforschung der Schlafkrankheit im Jahre* 1906–07 *nach Ostafrika entsandten Kommission.* Berlin.

KOCH, R., & GAFFKY, G. (1887). Bericht über die Thätigkeit der zur Erforschung der Cholera im Jahre 1883 nach Ägypten und Indien entsandten Kommission. *Arb. K. Gesundheitsamte*, III, 1.

KÖLLIKER, A. (1845). Die Lehre von der thierischen Zelle und den einfacheren thierischen Formelementen, nach den neuesten Fortschritten dargestellt. *Zeitschr. Wiss. Botan.*, I, ii, 46.

—— (1848). Beiträge zur Kenntniss niederer Thiere. I. Ueber die Gattung *Gregarina*. *Zeitschr. Wiss. Zool.*, I, 1.

—— (1854). *Mikroskopische Anatomie oder Gewebelehre des Menschen.* Leipzig

KOFOID, C. A. (1917). The biological and medical significance of the intestinal flagellates. *II Pan-Amer. Scient. Congr., Washington, December 27, 1915, to January 8, 1916*, 1.

—— (1920). A critical review of the nomenclature of human intestinal flagellates, *Cercomonas, Chilomastix, Trichomonas, Tetratrichomonas* and *Giardia. Univ. California Publ. Zool.*, XX. 145.

—— (1920a). Hookworm and amœbiasis in California. *California State Journ. Med.*, XVIII, 329.

—— (1921). Symposium on fertilization. 81. A critical discussion of the chromidial formation of nuclei, of autogamy, and of the multiple division of polyenergid nuclei, with especial reference to the unity of the mechanism of heredity in the Protozoa and Metazoa. *Anat. Rec.*, XX, 223.

—— (1923). Amœba and Man. *Univ. California Chron.*, p. 149.

KOFOID, C. A., BOECK, W. C., MINNICH, D. E., & ROGERS, J. H. (1919). On the treatment of giardiasis in rats with arsenobenzol. *Journ. Med. Res.*, XXXIX, 293.

KOFOID, C. A., BOYERS, L. M., & SWEZY, O. (1922). *Endamœba dysenteriæ* in the lymph glands of man in Hodgkin's disease. *Univ. California. Publ. Zool.*, XX, 309.

KOFOID, C. A., & CHRISTIANSEN, E. B. (1915). On *Giardia microti*, sp. nov., from the meadow mouse. *Ibid.*, XVI, 23.

KOFOID, C. A. & CHRISTIANSEN, E. B. (1915a). On binary and multiple fission in *Giardia muris* (Grassi). *Ibid.*, XVI, 30.

—— —— (1915b). On the life-history of *Giardia*. *Proc. Nat. Acad. Sci.*, Washington, I, 547.

KOFOID, C. A., KORNHAUSER, S. I., & PLATE, J. T. (1919). Intestinal parasites in overseas and home service troops of the U.S. army. *Journ. Amer. Med. Assoc.*, LXXII, 1721.

KOFOID, C. A., KORNHAUSER, S. I., & SWEZY, O. (1919). Criteria for distinguishing the *Endamœba* of amebiasis from other organisms. *Arch. Intern. Med.*, XXIV, 35.

—— —— —— (1919a). Structure and systematic relationships of the "iodine cysts" from human fæces. *Milit. Surg.*, XLV, 30.

KOFOID, C. A., & McCULLOCH, I. (1916). On *Trypanosoma triatomœ*, a new flagellate from a hemipteran bug from the nests of the wood rat, *Neotoma fuscipes*. *Univ. California Publ. Zool.*, XVI, 113.

KOFOID, C. A & SWEZY, O, (1915). Mitosis in *Trichomonas*. *Proc. Nat. Acad. Sci.*, Wash., I, 315.

—— —— (1915a). Mitosis and multiple fission in trichomonad flagellates. *Proc. Amer. Acad. Arts & Sci.*, LI, 289.

—— —— (1919). Studies on the parasites of the termites—I-IV. *Univ. Cal. Publ. Zool.*, XX, 1.

—— —— (1919a). Flagellate affinities of *Trichonympha*. *Proc. Nat. Acad. Sci.*, Wash., V, 9.

—— —— (1920). On the morphology and mitosis of *Chilomastix mesnili* (Wenyon), a common flagellate of the human intestine. *Univ. California Publ. Zool.*, XX, 117.

—— —— (1920a). On the prevalence of carriers of *Endamœba dysenteriœ* among soldiers returned from overseas service. *New Orleans Med. & Surg. Journ.*, LXXIII, 4.

—— —— (1921). *Councilmania lafleuri*, a new amœba of the human intestine. *Proc. Soc. Exp. Biol. & Med.*, XVIII, 310.

—— —— (1921a). On the free, encysted, and budding stages of *Councilmania lafleuri*, a parasitic amœba of the human intestine. *Univ. California Publ. Zool.*, XX, 169.

—— —— (1921b). The flagellate infections of the human digestive tract. Nelson's *Loose Leaf Encyclop. Living Med.*, V, 365.

—— —— (1921c). On the prevalence of carriers of *Endamœba dysenteriœ* among soldiers returned from overseas service. *Amer. Journ. Trop. Med.*, I, 41.

—— —— (1922). Mitosis and fission in the active and encysted phases of *Giardia enterica* (Grassi) of man, etc. *Univ. California Publ. Zool.*, XX, 199.

—— —— (1922a). Mitosis in *Endamœba dysenteriœ* in the bone marrow in arthritis deformans. *Ibid.*, XX, 301.

—— —— (1922b). *Endamœba dysenteriœ* in the lymph glands of man in Hodgkin's disease. *Ibid.*, XX, 309.

—— —— (1924). Pentatrichomoniasis in man. *Amer. Journ. Trop. Med.*. IV, 33.

—— —— (1924a). The cytology of *Endamœba gingivalis* (Gros) Brumpt compared with that of *E. dysenteriœ*, with special reference to the determination of the amœbas in bone marrow in arthritis deformans of Ely's second type. *Univ. California Publ. Zool.*, XXVI, 165.

—— —— (1924b). *Karyamœba falcata*, a new amœba from the human intestinal tract. *Ibid.*, XXVI, 221.

—— —— (1925). On the number of chromosomes and the type of mitosis in *Endamœba dysenteriœ*. *Ibid.*, XXVI, 331.

—— —— (1925a). *Karyamœbina* substituted for *Karyamœba*, with a note on its occurrence in man. *Ibid.*, XXVI, 435.

KOFOID, C. A., SWEZY, O., & KESSEL, J. F. (1923). On the genus *Councilmania*, budding intestinal amœbæ parasitic in man and rodents. *Ibid.*, XX, 431.

—— —— —— (1924). On the distinctions between *Entamœba coli* and *Councilmania lafleuri*. *Ibid.*, XXVI, 21.

KOFOID, C. A., & WAGENER, E. H. (1925). The behavior of *Endamœba dysenteriœ* in mixed cultures with bacteria. *Ibid.*, XXVIII, 127.

—— (1925a). Studies of the effects of certain drugs upon *Endamœba dysenteriœ in vitro*. *Ibid.*, XXVIII, 155.

KOHL-YAKIMOFF, N., & YAKIMOFF, W. L. (1915). Hämogregarinen der Seefische. *Centralbl. Bakt.*, I. Abt., LXXVI, 135.

KOHL-YAKIMOFF, N., YAKIMOFF, W. L., & SCHOKHOR, N. J. (1913). Leishmaniose canine à Taschkent. *Bull. Soc. Path. Exot.*, VI, 432.

KOIDZUMI, M. (1921). Studies on the intestinal Protozoa found in the termites of Japan. *Parasitology*, XIII, 235.

KOLLE, W. (1898). Ueber einen neuen pathogenen Parasiten im Blut der Rinder in Süd-Afrika. *Zeitschr. Hyg.- u. Infekt.*, XXVII, 45.

KONSULOFF, S. (1922). Untersuchungen über *Opalina*. *Arch. Protist.*, XLIV, 285.

KORKE, V. T. (1914). A note on the production of localized lesions by *Leishmania donovani* in *Macacus sinicus*. *Indian Journ. Med. Res.*, I, 622.

—— (1916). On a nosema (*Nosema pulicis*, n.s.) parasitic in the dog flea (*Ctenocephalus felis*). *Ibid.*, III, 725.

KOSSEL, H. (1899). Ueber einen malariaähnlichen Blutparasiten bei Affen. *Zeitschr. Hyg.- u. Infekt.*, XXXII, 25.

KOSSEL. H., WEBER, A., SCHUTZ, & MIESSNER (1904). Über die Hämoglobinurie der Rinder in Deutschland. *Arb. K. Gesundheitsamte*, XX, 1.

KOTLÁN, A. (1922). Giardien (Lamblien) in Vögeln. *Centralbl. Bakt.*, I Abt., LXXXVIII. 54.

—— (1923). Zur Kenntnis der Darmflagellaten aus der Hausente und anderen Wasservögeln. *Ibid.*, I. Abt., XC, 24.

KOVÁCS, F. (1892). Beobachtungen und Versuche über die sogenannte Amoebendysenterje. *Zeitschr. Heilk.*, XIII, 509.

KRASSILSTSCHIK, J. (1866). Ueber eine neue Flagellate *Cercobodo laciniœgerens*, n. g. et n. sp. *Zool. Anz.*, IX, 365, 394.

KRAUSE, P. (1906). Ueber Infusorien im Typhusstuhle nebst Beschreibung einer bisher noch nicht beobachteten Art (*Balantidium giganteum*). *Deutsch. Arch. Klin. Med.*, LXXXVI, 442.

KREMPF, A. (1917). Sur un hématozoaire endoglobulaire nouveau de l'homme (*Hœmogregarina hominis*). *C. R. Acad. Sci.*, CLXIV, 965.

KRUMBHAAR, E. B. (1918). Experimental trypanosomiasis: *T. equiperdum* infection in the dog. *Journ. Infect. Dis.*, XXII, 34.

KRUSE, W. (1890). Über Blutparasiten. *Arch. Path. Anat.*, CXX, 541; CXXI, 359.

KRUSE, W., & PASQUALE, A. (1894). Untersuchungen über Dysenterie und Leberabscess. *Zeitschr. Hyg.- u. Infekt.*, XVI, 1.

KUCZYNSKI, M. H. (1914). Untersuchungen an Trichomonaden. *Arch. Protist.*, XXXIII, 119.

— (1917). Über die Teilung der Trypanosomenzelle nebst Bemerkungen zur Organisation einiger nahestehender Flagellaten. *Ibid.*, XXXVIII, 94.

—— (1918). Ueber die Teilungsvorgänge verschiedener Trichomonaden und ihre Organisation im allgemeinen. *Ibid.*, XXXIX, 107.

KUDICKE, [R.] (1906). Ein Beitrag zur Kenntnis der menschlichen Trypanosomakrankheit. *Centralbl. Bakt.*, I. Abt., XLI, 72.

KUDO, R. (1916). On the structure and life-history of *Nosema bombycis* Nägeli. *Bull. Imp. Sericult. Exp. Stat.*, I, 31.

—— (1918). Contributions to the study of parasitic Protozoa. IV. Note on some Myxosporidia from certain fish in the vicinity of Woods Hole. *Journ. Parasit.*, V, 11.

—— (1919). Studies on Myxosporidia. Synopsis of genera and species of Myxosporidia. *Illinois Biol. Monogr.*, V, Nos. 3 & 4.'

—— (1920). On the structure of some microsporidian spores. *Journ. Parasit.*, VI, 178.

—— (1921). Studies on Microsporidia, with special reference to those parasitic in mosquitoes. *Journ. Morph.*, XXXV, 153.

—— (1922). On the Protozoa parasitic in frogs. *Trans. Amer. Micro. Soc.*, XLI, 59.

—— (1922a). Studies on Microsporidia parasitic in mosquitoes. *Journ. Parasit.*, VIII, 70.

—— (1922b). On the morphology and life-history of a Myxosporidian, *Leptotheca ohlmacheri*, parasitic in *Rana clamitans* and *R. pipiens*. *Parasitology*, XIV, 221.

—— (1924). Studies on Microscoporidia parasitic in mosquitoes. III. On *Thelohania legeri* Hesse (=*Th. illinoisensis* Kudo). *Arch. Protist.*, XLIX, 147.

—— (1924a). Studies on Microsporidia parasitic in mosquitoes. VI. On the development of *Thelohania opacita*, a culicine parasite." *Journ. Parasit.*, XI, 84.

—— (1924b). A biologic and taxonomic study of the Microsporidia. *Illinois Biol. Monogr.*, IX, Nos. 2 & 3, 1.

—— (1926). Observations on *Endamœba blattœ*. *Amer. Journ. Hyg.*, VI, 139.

KUDO, R., & HETHERINGTON, D. C. (1922). Notes on a microsporidian parasite of a nematode. *Journ. Parasit.*, VIII, 129.

KÜCHENMEISTER (1852). Beiträge zur Helminthologie, mit besonderer Rücksicht auf patholog. Anatomie, so wie über Leberkrankheiten der Kaninchen, Rinder u.s.w. *Arch. Path. Anat.*, IV, 83.

KÜHN, A. (1915). Über Bau, Teilung und Encystierung von *Bodo edax* Klebs. *Arch. Protist.*, XXXV, 212.

KÜHN, A., & SCHUCKMANN, W. v. (1912). Cytologische Studien an Trypanosomen. *Zool. Jahrb.*, XV, Suppl., 329.

KÜHN, J. (1865). Untersuchungen über die Trichinenkrankheit der Schweine. *Inst. d. Univ. Halle*, 68.

KÜLZ, L. (1916). Pathologische und therapeutische Beobachtungen aus Niedermesopotamien. *Arch. Schiffs.- u. Trop.-Hyg.*, XX, 487.

KUENEN, W. A., & SWELLENGREBEL, N. H. (1913). Die Entamoeben des Menschen und ihre praktische Bedeutung. *Centralbl. Bakt.*, I. Abt., LXXI, 378.

—— —— (1917). Korte beschrijving van enkele minder bekende protozoën uit den menschelijken darm. *Geneesk. Tijdschr. Nederl.-Indie*, LVII, 496.

KUMM, H. (1922). Zur Epidemiologie der Schafkokzidiose. *Berlin. Tierärztl. Woch.*, XXXVIII, 318.

KUNSTLER. J. (1882). Sur cinq protozoaires parasites nouveaux. *C. R. Acad. Sci.*, XCV, 347.

—— (1883). Recherches sur les infusoires parasites. Sur quinze protozoaires nouveaux. *Ibid.*, XCVII, 755.

—— (1884). *Trichomonas vaginalis* Don. *Journ. Microgr.*, VIII, 317.

KUNSTLER, J. (1898). Observations sur le *Trichomonas intestinalis*, Leuckart. *Bull. Sci. France & Belgique*, XXXI, 185.

KUNSTLER, J., & GINESTE, C. (1907). *Giardia alata* (nov. spec.). *C. R. Acad. Sci.*, CXLIV, 441.

KUNZE, W. (1907). Über *Orcheobius herpobdellæ* Schuberg et Kunze, ein Coccidium aus *Herpobdella atomaria* Car. (*Nephelis vulgaris* Moq.-Tand.). *Arch. Protist.*, IX, 382.

KUPKE, A. (1923). Untersuchungen über *Globidium leuckarti* Flesch. *Zeitschr. Infektionskr. d. Haust.*, XXIV, 210.

KUSAMA, S., KASAI, K., & KOBAYASHI, R. (1919). The leucocytogregarine of the wild rat, with special reference to its life-history. *Kitasato Arch.*, III, 103.

KUSCHAKEWITSCH, S. (1907). Beobachtungen über vegetative, degenerative und germinative Vorgänge bei den Gregarinen des Mehlwurmdarms. *Arch. Protist.*, Suppl. I, 202.

LABBÉ, A. (1893). Sur les coccidies des oiseaux. *C. R. Acad. Sci.*. CXVII. 407.

—— (1894). Recherches zoologiques et biologiques sur les parasites endoglobulaires du sang des vertébrés. *Arch. Zool. Exp.*, II, 55.

—— (1895). *Bananella lacazei* genre nouveau de coccidie oligosporée. *Ibid.*, III, (N. & R.), xv.

—— (1895a). Sur le noyau et la division nucléaire chez les *Benedenia*. *C. R. Acad. Sci.*, CXX, 381.

—— (1896). Recherches zoologiques, cytologiques et biologiques sur les coccidies. *Arch. Zool. Exp.*, IV. 517.

—— (1899). Sporozoa. (In *Das Tierreich*. Berlin.)

LABBÉ, M., TARGHETTA, & AMEUILIE (1918). Le kala-azar infantile en France. *Bull. Acad. Méd.*, LXXIX, 288.

LABERNADIE, V. G. F., & HUBAC (1923). Sur l'*Endotrypanum schaudinni* de l'unau, edenté de la Guyane (*Cholœpus didactylus*). *C. R. Soc. Biol.*, LXXXVIII, 664.

LA CAVA, F. (1912). Sulla leishmaniosi delle mucose e del primo reperto di *Leishmania tropica* flagellata nel corpo umano. *Malaria e Mal. Paesi Caldi*, III, 310.

—— (1914). Ueber Häufigkeit, Verbreitung und Symptome der Leishmaniose der Haut und der Schleimhäute in Unteritalien. Aeusserliche Leishmaniose. *Centralbl. Bakt.*, I. Abt., LXXIV, 494.

LAFONT, A. (1909). Sur la présence d'un parasite de la classe des flagellés dans le latex de l'*Euphorbia pilulifera*. *C. R. Soc. Biol.*, LXVI, 1011.

—— (1911). Sur la transmission du *Leptomonas davidi* des euphorbes par un hémiptere *Nysius euphorbiæ*. *Ibid.*, LXX, 58.

—— (1912). Trypanosomide d'un réduvide (*Conorhinus rubrofasciatus*) inoculable au rat et à la souris. *Ann. Inst. Pasteur*, XXVI, 893.

LAFONT, A., & HECKENROTH, F. (1915). Un cas de leishmaniose canine à Dakar. *Bull. Soc. Path. Exot.*, VIII, 162.

LAGRANGE, E. (1922). Contribution à l'étude des piroplasmes des bovidés. *Ibid.*, XV, 295.

LAMBL, W. (1859). Mikroskopische Untersuchungen der Darmexkrete. Beitrag zur Pathologie des Darms und zur Diagnostik am Krankenbette. *Vierteljahrsschr. Prakt. Heilk.*, LXI, 1.

—— (1860). Beobachtungen und Studien aus dem Gebiete der pathologischen Anatomie und Histologie (Löschner u. Lambl.). *Fr. Jos.-Kind.-Spit. Prag.*, I. Th., Sec. XIII (6) '' Zur Pathologie des Darms,'' 365.

LAMBORN, W. A. (1920). Sleeping sickness—Marimba. *Ann. Med. Rept. Health San. Condit. Nyasaland Protect.*, *1919*, Append., 48.

—— (1921). A Protozoon pathogenic to mosquito larvæ. *Parasitology*, XIII, 213.

LANDIS, E. M. (1920). An amicronucleate race of *Paramœcium caudatum*. *Amer. Natural.*, LIV, 453.

LANFRANCHI, A. (1908). Di una speciale forma morbosa nei piccioni dovuta ad un emotozoario della famiglia mastigofori. *Moderno Zooiatro.*, Suppl. to No. 6, 289.

—— (1915). Sur le passage des trypanosomes dans le lait. *Bull. Soc. Path. Exot.*, VIII, 438.

—— (1916). Sul possibile passagio dei tripanosomi nel latte. *Atti R. Accad. Lincei, Rendic.*, XXV, i, 369.

—— (1917). Su di un nuovo genere di parassiti del sangue. *Annali d'Igiene*, XXVII, 697.

—— (1918). Sul possibile passagio dei tripanosomi nel latte. *Atti R. Accad. Lincei, Rendic.*, XXVII, i, 62.

—— (1918a). Sur la possibilité du passage des trypanosomes dans le lait. *Arch. Ital. Biol.*, LXVIII, 158.

LANG, G. (1868). Ueber die Entstehungsweise der sogenannten Wurmknoten in der Leber. *Arch. Path. Anat.*, XLIV, 202.

LANGERON, M. (1911). Hématies en demi-lune dans le sang du rat et du cobaye. *C. R. Soc. Biol.*, LXX, 434.

—— (1920). Sur une hémogrégarine d'une macaque. *Bull. Soc. Path. Exot.*, XIII, 165.

—— (1920a). Note additionnelle sur une hémogrégarine d'un macaque. *Ibid.*, XIII, 394.

LANKESTER, E. R. (1870). Remarks on *Opalina* and its contractile vesicles, on pachydermon and annelidan spermatophors. *Quart. Journ. Micro. Sci.*, X, 143.

LANKESTER, E. R. (1871). On *Undulina*, the type of a new group of infusoria. *Ibid.*, XI, 387.

—— (1882). On *Drepanidium ranarum* the cell parasite of the frog's blood and spleen (Gaule's Würmchen). *Ibid.*, XXII, 53.

LARROUSSE, F. (1921). *Etude systématique et médicale des phlébotomes.* Paris.

LAUTERBORN, R. (1908). Protozoen-Studien. V. Tl. Zur Kenntnis einiger Rhizopoden und Infusorien aus dem Gebiete des Oberrheins. *Zeitschr. Wiss. Zool.*, XC, 645.

LAVERAN, A. (1880). Note sur un nouveau parasite trouvé dans le sang de plusieurs malades atteints de fièvre palustre. *Bull. Acad. Méd.*, IX, 1235, 1268, 1346.

—— (1880a). Un nouveau parasite trouvé dans le sang des malades atteints de fièvre palustre. *Bull. & Mém. Soc. Méd. Hôpit. Paris*, XVII, 158.

—— (1880b). Contribution à l'étude du bouton de Biskra. *Ann. Dermat. & Syph.*, I, 173.

—— (1884). *Traité des fièvres palustres*, Paris, 457.

—— (1891). Sur des hématozoaires de l'alouette voisins de ceux du paludisme. *C. R. Soc. Biol.*, XLIII, 383.

— — (1898). Sur les modes de reproduction de *Klossia helicina* (Schneider). *Ibid.*, L, 1083.

— - - (1898a). Sur les modes de reproduction d'*Isospora lacazei*. *Ibid.*, L, 1139.

—— (1899). Les hématozoaires endoglobulaires (Hæmocytozoa). *Cinq. d. l. Soc. Biol.*, vol. jub., 124.

—— (1899a). Sur le bacille parasite des hématies de *Rana esculenta*. *C. R. Soc. Biol.*, LI, 355.

—— (1900). Au sujet de l'hématozoaire endoglobulaire de *Padda orizivora*. *Ibid.*, LII, 19.

—— (1901). Contribution à l'étude du *Piroplasma equi*. *Ibid.*, LIII, 385.

—— (1902). Sur une *Hæmamæba* d'une désange (*Parus major*). *Ibid.*, LIV, 1121.

—— (1902a). Sur un nouveau trypanosome des bovidés (*Trypanosoma theileri*). *C. R. Acad. Sci.*, CXXXIV, 512.

—— (1902b). De l'action du sérum humain sur le trypanosome du nagana (*Tr. brucei*). *Ibid.*, CXXXIV, 735.

- — (1902c). Sur quelques hémogrégarines des ophidiens. *Ibid.*, CXXXV, 1036.

—— (1903). De l'action de sérum humain sur les trypanosomes du nagana, du caderas et du surra. *Ibid.*, CXXXVII, 15.

- —— (1904). Action du sérum humain sur quelques trypanosomes pathogènes; action de l'acide arsénieux sur *Tr. gambiense*. *Ibid.*, CXXXVIII, 450.

—— (1904a). Immunité naturelle des cynocéphales pour les trypanosomiases, activité de leur sérum sur les trypanosomes. *Ibid.*, CXXXIX, 177.

- — (1904b). Trypanoplasme et trypanosome du vairon. *C. R. Soc. Biol.*, LVII, 250.

—— (1904c). Sur deux mémoires de M. Cazalbou, ayant pour titres. 1°. Mbori expérimentale, et 2°. Note sur la soumaya. *Bull. Acad. Méd.*, LI, 348.

- —— (1905). Sur une hémamibe nouvelle de *Testudo pardalis*. *C. R. Soc. Biol.*, LIX, 176.

- - - (1905a). Trypanosomiases et tsétsé dans la Guinée française. *C. R. Acad. Sci.*, CXL, 75.

—— (1905b). Hæmocytozoa. Essai de classification. *Bull. Inst. Pasteur*, III, 809.

—— (1905c). Sur une hémogrégarine des gerboises. *C. R. Acad. Sci.*, CXLI, 295.

·—— (1905d). Note pour servir à l'histoire des trypanosomiases du Soudan anglo-égyptien. *C. R. Soc. Biol.*, LVIII, 292.

—— (1906). Trypanosomiases du Haut-Niger; un nouveau trypanosome pathogène. *C. R. Acad. Sci.*, CXLIII, 94.

—— (1907). Sur les trypanosomiases du Haut-Niger. *Ann. Inst. Pasteur*, XXI, 321.

—— (1911). Identification et essai de classification des trypanosomes de mammifères. *Ibid.*, XXV, 497.

·—— (1911a). Au sujet de *Trypanosoma rhodesiense* (Stephens et Fantham). *C. R. Acad. Sci.*, CLIII, 1112.

—— (1912). Au sujet de *Trypanosoma rhodesiense* (Stephens et Fantham). *Bull. Soc. Path. Exot.*, V, 26.

·—— (1912a). Expériences d'immunité croisée avec *Trypanosoma brucei*. *Tr. brucei* var. *werbitzkii* et *Tr. rhodesiense*. *Ibid.*, V, 101.

—— (1912b). Contribution à l'étude des infections expérimentales produites par le *Trypanosoma rhodesiense*. *Ibid.*, V, 241.

—— (1912c). Infections des souris et des rats dues au kala-azar méditerranéen et au kala-azar indien. *Ibid.*, V, 715.

—— (1912d). Présentation de macaques inoculés avec succès au moyen d'une culture de la *Leishmania* du bouton de Delhi. *Ibid.*, V, 573.

—— (1913). Les macaques et les chiens sont sensibles au kala-azar indien comme au kala-azar méditerranéen. *C. R. Acad. Sci.*, CLVII, 898.

—— (1914). [Remarks on paper by Emin (1914).] *Bull. Soc. Path. Exot.*, VII, 387.

—— (1914a). Nouveaux faits tendant à démontrer que le kala-azar méditerranée doit être identifié au kala-azar indien. *C. R. Acad. Sci.*, CLVIII, 1060.

·—— (1914b). Infections expérimentales de souris, d'un meriones, d'un rat et d'un macaque par la *Leishmania tropica*. *Bull. Soc. Path. Exot.*, VII, 663.

LAVERAN, A. (1915). Des lacertiens peuvent-ils être infectés par des Leishmania ? *Ibid.*, VIII, 104.

—— (1915a). Au sujet d'un *Trypanosoma gambiense* qui, conservé depuis 12 ans chez des animaux, est resté résistant au sérum humain. *Ibid.*, VIII, 442.

—— (1915b). Nouvelle contribution à l'étude des infections expérimentales de la souris par *Leishmania tropica*. *Ibid.*, VIII, 363.

—— (1915c). Nouvelle contribution à l'étude des infections expérimentales de la souris par la *Leishmania tropica*, un cas d'infection de la gerbille. *Ibid.*, VIII, 680.

—— (1915d). Les leishmanioses chez les animaux. *Ann. Inst. Pasteur*, XXIX, 71.

—— (1916). Leishmaniose cutanée expérimentale chez les macaques et chez le chien. Conditions de l'immunité. *Bull. Soc. Path. Exot.*, IX, 265.

—— (1917). *Leishmanioses*. Paris.

—— (1920). La leishmaniose canine chez la souris. *Bull. Soc. Path. Exot.*, XIII, 680.

LAVERAN, A., & FRANCHINI, G. (1913). Infezione sperimentale del topo bianco coll'*Herpetomonas ctenocephali*. *Pathologica*, V, 605. (French version *C. R. Acad. Sci.*, CLVII, 423.)

—— —— (1913a). Infections expérimentales de mammifères par des flagellés du tube digestif de *Ctenocephalus canis* et d'*Anopheles maculipennis*. *C. R. Acad. Sci.*, CLVI, 744.

—— —— (1913b). *Trypanosoma talpæ* chez *Palæcpsylla gracilis*. *C. R. Soc. Biol.*, LXXIV, 1254.

—— —— (1914). Contribution à l'étude des "marginal points" des hématies de mammifères. *Bull. Soc. Path. Exot.*, VII, 580.

—— —— (1914a). Infections de mammifères par des flagellés d'invertebrés. *Ibid.*, VII, 605.

—— —— (1914b). Infection naturelle du rat et de la souris au moyen de puces de rat parasitées par *Herpetomonas pattoni*. *C. R. Acad. Sci.*, CLVIII, 450.

—— —— (1914c). Infection de la souris au moyen des flagellés de la puce du rat, par la voie digestive. *Ibid.*, CLVIII, 770.

—— —— (1915). Au sujet d'un herpetomonas de *Ctenopsylla musculi* et de sa culture. *Bull. Soc. Path. Exot.*, VIII, 266.

—— —— (1919). Infection des souris blanches à l'aide des cultures de *Herpetomonas ctenocephali*. *Ibid.*, XII, 379.

—— —— (1919a). Sur les flagellés parasites de quelques insectes et sur les infections qu'ils peuvent produire chez les souris. *Ibid.*, XII, 665.

—— —— (1920). Infections expérimentales de chiens et de cobayes à l'aide de cultures d'*Herpetomonas* d'insectes. *Ibid.*, XIII, 569.

—— —— (1920a). Contribution à l'étude de la flagellose des euphorbes. *Ibid.*, XIII, 796.

—— —— (1920b). Contribution à l'étude des flagellés des culicides, des muscides, des phlébotomes et de la blatte orientale. *Ibid.*, XIII, 138.

—— —— (1921). Sur un *Herpetomonas* du loir. *Ibid.*, XIV, 278.

—— —— (1921a). Des hématozoaires du gecko et spécialement de *Herpetomonas tarentolæ*. Procédé simple de culture des *Herpetomonas*. *Ibid.*, XIV, 323.

—— —— (1923). Expériences sur les flagellés de la punaise du chou. *Ibid.*, XVI, 319.

LAVERAN, A., & LUCET (1905). Deux hématozoaires de la perdrix et du dindon. *C. R. Acad. Sci.*, CXLI, 673.

LAVERAN, A., & MARULLAZ, M. (1914). Sur deux hémamibes et un toxoplasme du *Liothrix luteus*. *Bull. Soc. Path. Exot.*, VII, 21.

—— —— (1914a). Sur la nature des corps de Graham-Smith. *Ibid.*, VII, 240.

LAVERAN, A., & MESNIL, F. (1899). De la sarcocystine, toxine des sarcosporidies. *C. R. Soc. Biol.*, LI, 311.

—— —— (1900). Sur une myxosporidie des voies biliaries de l'hippocampe. *Ibid.*, LII, 380.

—— —— (1900a). Sur l'agglutination des trypanosomes du rat par divers sérums. *Ibid.*, LII, 939.

—— —— (1901). Deux hémogrégarines nouvelles des poissons. *C. R. Acad. Sci.*, CXXXIII, 572.

—— —— (1901a). Recherches morphologiques et experimentales sur le trypanosome des rats (*Tr. lewisi* Kent). *Ann. Inst. Pasteur*, XV, 673.

—— —— (1901b). Sur la morphologie et la systématique des flagellés à membrane ondulante (genres *Trypanosoma* Gruby et *Trichomonas* Donné). *C. R. Acad. Sci.*, CXXXIII, 131.

—— —— (1901c). Sur les flagellés à membrane ondulante des poissons (genres *Trypanosoma* Gruby et *Trypanoplasma*, n. gen.). *Ibid.*, CXXXIII, 670.

—— —— (1901d). Sur la structure du trypanosome des grenouilles et sur l'extension du genre *Trypanosoma* Gruby. *C. R. Soc. Biol.*, LIII, 678.

—— —— (1902). Sur quelques protozoaires parasites d'une tortue d'Asie (*Damonia reevesii*). *C. R. Acad. Sci.*, CXXXV, 609.

—— —— (1902a). Sur la multiplication endogène des myxosporidies. *C. R. Soc. Biol.*, LIV, 469.

—— —— (1902b). Sur deux coccidies intestinales de la *Rana esculenta*. *Ibid.*, LIV, 857.

—— —— (1902c). Sur la coccidie trouvée dans le rein de la *Rana esculenta* et sur l'infection générale qu'elle produit. *C. R. Acad. Sci.*, CXXXV, 82.

—— —— (1903). [Demonstration by Laveran.] *Bull. Acad. Méd.*, L, 238.

—— —— (1903a). Sur un protozoaire nouveau (*Piroplasma donovani* Lav. et Mesn.), parasite d'une fièvre de l'Inde. *C. R. Acad. Sci.*, CXXXVII, 957.

LAVERAN, A. & MESNIL, F. (1904). *Trypanosomes et trypanosomiases*. Paris.
—— (1912). *Trypanosomes et trypanosomiases*. 2nd edition. Paris.
LAVERAN, A., & NATTAN-LARRIER (1912). Au sujet de *Trypanosoma rhodesiense* (Stephens et Fantham). *C. R. Acad. Sci.*, CLIV, 18.
—— —— (1912a). Contribution à l'étude de la espundia. *Bull. Soc. Path. Exot.*, V, 176, 487.
—— —— (1912b). Séro-diagnostic des infections à *Trypanosoma gambiense* et à *Tr. rhodesiense*. *Ibid.*, V, 220.
—— —— (1912c). Le *Trypanosoma rhodesiense*, devenu résistant au sérum humain, perd assez facilement cette propriété. *Ibid.*, V, 367.
—— —— (1913). Piroplasmoses canines d'Europe et d'Afrique. *Ann. Inst. Pasteur*, XXVII, 701.
LAVERAN, A., & NÈGRE (1905). Sur un protozoaire parasite de *Hyalomma ægyptium*. *C. R. Soc. Biol.*, LVIII, 964.
LAVERAN, A., & NICOLLE, M. (1899). Contribution à l'étude de *Pyrosoma bigeminum*. *Ibid.*, LI, 748.
—— —— (1899a). Hématozoaires endoglobulaires du mouton. *Ibid.*, LI, 800.
LAVERAN, A., & PETTIT, A. (1909). Sur une hémogrégarine de *Pituophis melanoleucus*. *C. R. Acad. Sci.*, CXLIX, 94.
—— —— (1909a). Infections légères du rat et de la souris par la *Leishmania donovani*. *C. R. Soc. Biol.*, LXVI, 911.
—— —— (1909b). Infection légère du cobaye par la *Leishmania donovani*. *Ibid.*, LXVII, 8.
—— —— (1909c). Contribution à l'étude des hémogrégarines de " *Lacerta ocellata* " Daud. *Bull. Soc. Path. Exot.*, II, 295.
LAVERAN, A., & VALLÉE (1905). Sur un cas de transmission par des ixodes de la spirillose et de la piroplasmose bovines. *C. R. Acad. Sci.*, CXL, 1515.
LAVIER, G. (1921). Les parasites des invertébrés hématophages. *Trav. Lab. Parasit. Fac. Méd. Paris.*
—— (1921a). Hémogrégarines, *Grahamella*, spirochète et trypanosome du campagnol indigène *Microtus arvalis* Pallas. *Bull. Soc. Path. Exot.*, XIV, 569.
—— (1921b). Flagellés parasites intestinaux du campagnol indigène *Microtus arvalis* Pallas. *Ibid.*, XIV, 710.
—— (1923). Sur deux espèces nouvelles du genre *Giardia* : *G. viscaciæ* de la viscache (*Viscacia viscacia*) et *G. varani* du varan (*Varanus niloticus*). *Ann. Parasit. Hum. & Comp.*, I, 146.
—— (1924). Deux espèces de *Giardia* du rat d'égout parisien (*Epimys norvegicus*). *Ibid.*, II, 161.
—— (1924a). *Eimeria hessei* n. sp., coccidie intestinale de *Rhinolophus hipposideros*. *Ibid.*, II, 335.
LAVIER, G., & GALLIARD, H. (1925). Parasitisme sanguin d'un *Hexamitus* chez un crapaud *Bufo calamita*. *Ibid.*, III, 113.
LAWSON, M. R. (1911). The æstivo-autumnal parasite; its sexual cycle in the circulating blood of man, with a description of the morphological and biological characteristics of the parasite. *Journ. Exp. Med.*, XIII, 263.
—— (1912). The cause of malarial anemia and the intravascular migrations of the malarial parasite. *Arch. Intern. Med.*, IX, 420.
—— (1913). The extracellular relation of the malarial parasite to the red corpuscle, and its method of securing attachment to the external surface of the red corpuscle. *Journ. Exp. Med.*, XVII, 324.
—— (1914). A stage in the migration of the adult tertian malarial parasite. Evidence of the extracellular relation of the parasite to the red corpuscle. *Ibid.*, XIX, 450.
—— (1914a). Free malarial parasites, and the effect of migration of the parasites of tertian malarial infections. *Ibid.*, XIX, 523.
—— (1915). Adult tertian malarial parasites attached to peripheral corpuscular mounds. The extracellular relation of the parasites to the red corpuscles. *Ibid.*, XXI, 584.
—— (1916). Distortion of the malarial parasite. An interpretation of *Plasmodium tenue* (Stephens). *Ibid.*, XXIV, 291.
—— (1918). Æstivo-autumnal parasites. Multiple infection of red corpuscles and the various hypotheses concerning it. *Ibid.*, XXVII, 749.
—— (1919). Migration of parasites as the cause of anemia in æstivo-autumnal malarial infections. *Ibid.*, XXIX, 361.
LEBAILLY, C. (1906). Recherches sur les hématozoaires parasites des téléostéens marins. *Arch. Parasit.*, X, 348.
LEBEDEFF, W. (1910). Über *Trypanosoma rotatorium* (Gruby). *Festschr. f. R. Hertwig*, Jena, I, 397.
LEBEDEFF, W., & TSCHARNOTZKY, A. (1911). Ein neuer Parasit im Blute des Iltis *Microsoma mustelæ*. *Centralbl. Bakt.*, I. Abt., LVIII, 625.
LEBLANC, P. (1900). Parasites endoglobulaires du chien. Nature de l'ictère infectieux du chien. *C. R. Soc. Biol.*, LII, 70.

LEBLANC, P. (1901). [Discussion following paper by Almy.] *Bull. Soc. Centr. Méd. Vét.*, LV, 380.

LEBŒUF, A. (1911). De la préparation de races de trypanosomes résistantes au sérum de cyno-céphales et au sérum humain. *Ann. Inst. Pasteur.*, XXV, 882.

—— (1921). Megalosplenie et hémogrégarine en Afrique équatoriale. (Extrait d'un rapport annuel du Médecin-major de 1re classe Lebœuf.) *Ann. Méd. & Pharm. Colon.*, XIX, 116.

LEBŒUF, A., & RINGENBACH (1910). Sur quelques hématozoaires du Congo (trypanosomes, microfilaires, leucocytozoaire). *Ann. Inst. Pasteur*, XXIV, 945.

LECLERQ, E. (1890). Les micro-organismes intermédiaires aux deux règnes. *Bull. Soc. Belge Micr.*, XVI, 70.

LEESE, A. S. (1909). Experiments regarding the natural transmission of surra carried out at Mohand in 1908. *Journ. Trop. Vet. Sci.*, IV, 107.

—— (1912). Biting flies and surra. *Ibid.*, VII, 19.

LEEUWENHOEK, A. v. (1722). De generatione Ranarum, etc. *Opera omnia*, 49.

LEGER. A. (1912). Leucocytozoaire de l'hyène tachetée du Haut-Sénégal et Niger. *C. R. Soc. Biol.*, LXXII, 1060.

—— (1912a). Présence de deux leucocytozoaires morphologiquement distincts dans le sang du chien à Bamako (Haut-Sénégal et Niger). *Ibid.*, LXXIII. 376.

—— (1922). Corps de Graham-Smith dans les hématies d'un primate (*Macacus rhesus*). *Bull. Soc. Path. Exot.*. XV, 679.

LEGER, A. & M. (1914). Sur un *Plasmodium* de la roussette du Haut-Sénégal et Niger. *C. R. Soc. Biol.*, LXXVII, 399.

—— (1920). Piroplasme de la civette au Sénégal. *Bull. Soc. Path. Exot.*, XIII, 649.

LÉGER, L. (1897). Coccidies nouvelles du tube digestif des myriapodes. *C. R. Acad. Sci.*, CXXIV, 901.

—— (1897a). Sur une nouvelle myxosporidie de la famille des Glugcidées. *Ibid.*, CXXV, 260.

—— (1897b). Sur la présence des coccidies chez les mollusques lamellibranches. *C. R. Soc. Biol.*, XLIX, 987.

—— (1897c). *Echinospora labbei*, nouvelle coccidie polysporée du tube digestif des Myriapodes. *Ibid.*, XLIX, 1082.

—— (1898). Essai sur la classification des coccidies et description de quelques espèces nouvelles ou peu connues. *Ann. Mus. Hist. Nat.*, Marseilles, I, 71.

—— (1900). Sur la présence d'une coccidie cœlomique chez *Olocrates abbreviatus*, Ol. *Arch. Zool. Exp.*, VIII (N. & R.), i.

—— (1900a). Sur un nouveau sporozoaire des larves de diptères (Schyzocystis). *C. R. Acad. Sci.*, CXXXI, 722.

—— (1900b). Le genre *Eimeria* et la classification des coccidies. *C. R. Soc. Biol.*. LII, 576.

—— (1902). Note sur le développement des éléments sexuels et la fécondation chez le *Stylorhynchus longicollis*, F. St. *Arch. Zool. Exp.*, X (N. & R.), lxix.

—— (1902a). Sur un flagellé parasite de l'*Anopheles maculipennis*. *C. R. Soc. Biol.*, LIV, 354.

—— (1903). Sur quelques cercomonadines nouvelles ou peu connues parasites de l'intestin des insectes. (Note préliminaire.) *Arch. Protist.*, II, 180.

—— (1904). Sporozoaires parasites de l'*Embia solieri* Rambur. *Ibid.*, III, 358.

—— (1904a). Sur la sporulation du *Triactinomyxon*. *C. R. Soc. Biol.*. LVI, 844.

—— (1904b). Considérations sur le genre *Triactinomyxon* et les actinomyxidies. *Ibid.*, LVI, 846.

—— (1904c). Sur un nouveau flagellé parasite des tabanides. *Ibid.*, LVII, 613.

—— (1904d). Protozoaires parasites des vipères. *Bull. Assoc. Franç. Avanc. Sci.*, IX, 268.

—— (1904e). Sur les hémoflagellés du *Cobitis barbatulæ* L. I. *Trypanosoma barbatulæ* n. sp. *C. R. Soc. Biol.*, LVII, 344.

—— (1904f). Sur la morphologie du *Trypanoplasma* des vairons. *C. R. Acad. Sci.*, CXXXVIII, 824.

—— (1904g). Sur les affinités de l'*Herpetomonas subulata* et la phylogénie des trypanosomes. *C. R. Soc. Biol.*, LVII, 615.

—— (1904h). La reproduction sexuée chez les *Stylorhynchus*. *Arch. Protist.*, III, 303.

—— (1905). Sur la présence d'un *Trypanoplasma* intestinal chez les poissons. *C. R. Soc. Biol.*, LVIII, 511.

—— (1907). Les schizogrégarines des trachéates. I. Le genre *Ophryocystis*. *Arch. Protist.*, VIII, 159.

—— (1909). Les schizogrégarines des trachéates. II. Le genre *Schizocystis*. *Ibid.*, XVIII, 83.

—— (1911). *Caryospora simplex*, coccidie monosporée et la classification des coccidies. *Ibid.*, XXII, 71.

LÉGER, L., & DUBOSCQ, O. (1902). Sur l'*Adelea dimidiata coccidioides* Léger et Duboscq. Coccidie parasite de la *Scolopendra oraniensis lusitanica* Verh. *C. R. Assoc. Franç. Avanc. Sci.*, XXXI, i, 230.

—— —— (1902a). Les grégarines et l'épithélium intestinal chez les trachéates. *Arch. Parasit.*, VI, 377.

Léger, L. & Duboscq, O. (1903). Recherches sur les myriapodes de Corse et leurs parasites. *Arch. Zool. Exp.*, I, 307.

—— —— (1903a). La reproduction sexuée chez *Pterocephalus*. *Ibid.*, I (N. & R.), cxli.

—— —— (1904). Notes sur les infusoires endoparasites. *Ibid.*, II, 337.

—— —— (1904a). Notes sur les infusoires endoparasites. 1. Les *Astomata* representent-ils un groupe naturel ? *Ibid.*, II (N. & R.), xcviii.

—— —— (1906). L'évolution d'une *Aggregata* de la seiche chez le *Portunus depurator* Leach. *C. R. Soc. Biol.*, LX, 1001.

—— —— (1908). L'évolution schizogonique de l'*Aggregata* (*Eucoccidium*) *eberthi* (Labbé). *Arch. Protist.*, XII, 44.

—— —— (1909). Protozoaires parasites de l'intestin du homard. *C. R. Acad. Sci.*, CXLVIII, 363; *Ann. Univ. Grénoble*, XXI, No. 1.

—— —— (1909a). Protistes parasites d'une larve de *Ptychoptera* et leur action sur l'hôte. *Bull. Acad. R. Belgique*, No. 8, 831, 885.

—— —— (1909b). Etudes sur la sexualité chez les grégarines. *Arch. Protist.*, XVII, 19.

—— —— (1910). *Selenococcidium intermedium* Lég. et Dub. et la systématique des sporozoaires. *Arch. Zool. Exp.*, XLV, 187.

—— —— (1911). *Spirocystis nidula* n. g., n. sp. *Bull. Soc. Zool.*, Paris, XXXVI, 62.

—— —— (1913). Le cycle évolutif de *Porospora portunidarum* Frenzel. *C. R. Acad. Sci.*, CLVI, 1932.

—— —— (1913a). Sur les premiers stades du développement des grégarines du genre *Porospora* (= *Nematopsis*). *C. R. Soc. Biol.*, LXXV, 95.

—— —— (1914). Sur une nouvelle schizogrégarine à stades épidermiques et à spores monozoiques. *Ibid.*, LXXVI, 296.

—— —— (1915). Etude sur *Spirocystis nidula* Lég. & Dub. schizogrégarine du *Lumbriculus variegatus* Müll. *Arch. Protist.*, XXXV, 199.

—— —— (1915a). *Pseudoklossia glomerata* n. g., n. sp. Coccidie de Lamellibranche. *Arch. Zool. Exp.*, LV (N. &. R.), 7.

—— —— (1917). *Pseudoklossia pectinis* n. sp. et l'origine des adéléidées. *Ibid.*, LVI (N. & R.), 88.

Léger, L., & Hesse, E. (1910). Cnidosporidies des larves d'ephémères. *C. R. Acad. Sci.*, CL, 411.

—— —— (1916). *Mrazekia*, genre nouveau de microsporidies à spores tubuleuses. *C. R. Soc. Biol.*, LXXIX, 345.

—— —— (1916a). Sur la structure de la spore des microsporidies. *Ibid.*, LXXIX, 1049.

—— —— (1921). Microsporidies à spores sphériques. *C. R. Acad. Sci.*, CLXXIII, 1419.

—— —— (1922). Coccidies d'oiseaux palustres. Le genre *Jarrina* n. g. *Ibid.*, CLXXIV, 74.

—— —— (1922a). Microsporidies bactériformes et essai de systématique du groupe. *Ibid.*, CLXXIV, 327.

Léger, L., & Hollande, A. C. (1912). La reproduction sexuée chez les coccidies monosporées du genre *Pfeifferinella*. *Arch. Zool. Exp.*, IX (N. &. R.), i.

Leger, M. (1912). Présence de *Hæmogregarina canis* en Corse. *C. R. Soc. Biol.*, LXXIII, 617.

—— (1918). Epizootie chez le cobaye paraissant due à une amibiase intestinale. *Bull. Soc. Path. Exot.*, XI, 163.

—— —— (1918a). Flagellés intestinaux de l'homme à la Guyane. *Ibid.*, XI, 368.

—— —— (1918b). Infection sanguine par *Leptomonas* chez un saurien. *C. R. Soc. Biol.*, LXXXI, 772.

—— —— (1920). Pyrexie mortelle à allure spéciale, causée par un flagellé à la Guyane française. *Ann. Inst. Pasteur.*, XXXIV, 481.

—— (1922). *Plasmodium* d'un singe de la Guinée française *Cercopithecus campbelli* Wath. *C. R. Soc. Biol.*, LXXXVI, 837.

—— (1922a). Formes crithidiennes observées chez *Lyperosia thirouxi* Roubaud. *Ibid.*, LXXXVII, 134.

Leger, M., & Baury, A. (1922). Trypanosome de l'écureuil fossoyeur du Sénégal. *Ibid.*, LXXXVII, 133.

—— (1923). Trypanosome de la chauve-souris du Sénégal *Hipposiderus tridens* Et. Geoff. *Ibid.*, LXXXVIII, 866.

Leger, M., & Bédier, E. (1922). Hémogrégarine du cynocéphale, *Papio sphinx* E. Geoffrey. *Ibid.*, LXXXVII, 933.

—— —— (1922a). Piroplasme du renard d'Afrique *Fennecus dorsalis* Gray. *Ibid.*, LXXXVII, 934.

—— —— (1922b). *Plasmodium* du lérot, " *Myoxus murinus* " Desmarest. *Ibid.*, LXXXVII, 1336.

—— —— (1923). *Plasmodium* d'un mustélidé, *Ictonyx zorilla*, du Sénégal. *Ibid.*, LXXXVIII, 422.

—— —— (1923a). Piroplasme d'un muridé. *Golunda campanæ*, du Sénégal. *Ibid.*, LXXXVIII, 424.

Leger, M., & Bouilliez, M. (1912). Sur un *Plasmodium* des singes. Passages par espèces variées. Action pathogène. *Ibid.*, LXXIII, 310.

LEGER, M. & BOUILLIEZ, M. (1913). Recherches expérimentales sur *Plasmodium inui* Halber-städter et Prowazek d'un *Macacus cynomolgus*. *Ann. Inst. Pasteur*, XXVII, 955.

LEGER, M., & MOUZELS, P. (1917). *Plasmodium de Iguana nudicollis*. *Bull. Soc. Path. Exot.*, X, 95.

—— —— (1917a). Piroplasme et microfiliare d'un édenté, le *Bradypus tridactylus* Linné. *Ibid.*, X, 173.

—— —— (1917b). Hémogrégarine intraleucocytaire d'un saurien, *Tupinambis nigropunctatus*. *Ibid.*, X, 283.

LEGER, M., & PORRY, E. (1918). Trypanosomes nouveaux de deux singes de la Guyane française. *C. R. Soc. Biol.*, LXXXI, 180.

LEGER, M., & TEJERA, E. (1920). Contribution à l'étude du *Trypanosoma venezuelense*, Mesnil, 1910. *Bull. Soc. Path. Exot.*, XIII, 576.

LEGER, M., & VIENNE, M. (1919). Epizootie à trypanosomes chez les bovidés de la Guyane française. *Ibid.*, XII, 258.

LEGRAS, S. (1918). Sur des formes hémogrégariniennes vues dans le sang et dans le tissu ganglion-naire des bovidés d'Algérie. *Ibid.*, XI, 274.

LEGROUX & LWOFF (1924). Présentation de préparations de sang de Padda montrant la parthéno-genèse des macrogamètes d'*Haemoproteus orizivorae*. *Ibid.*, XVII, 287.

LEIDY, J. (1846). Description of a new genus and species of Entozoa. *Proc. Acad. Nat. Sci.*, Philadelphia, III, 100.

—— (1847). [Name *Cryptobia* changed to *Cryptoicus*.] *Ibid.*, III, 239.

—— (1849). [No title to paper. Indexed as " On the existence of Entophyta in healthy animals as a natural condition."] *Ibid.*, IV, 233.

—— (1850). Two new species of infusorial Entozoa. *Ibid.*, V, 100.

—— (1851). Corrections and additions to former papers on Helminthology published in the Pro-ceedings of the Academy. *Ibid.*, V, 284.

—— (1855). Contributions towards a knowledge of the marine invertebrate fauna of Rhode Island and New Jersey. *Journ. Acad. Nat. Sci.*, Philadelphia, III, 135.

—— (1856). A synopsis of Entozoa and some of their ecto-congeners observed by the author. *Proc. Acad. Nat. Sci.*, Philadelphia, VIII, 42.

—— (1879). On *Amoeba blattae*. *Ibid.*, XXXI, 204.

LEISHMAN, W. B. (1903). On the possibility of the occurrence of trypanosomiasis in India. *Brit. Med. Journ.*, I, 1252.

LEISHMAN, W. B., & STATHAM, J. C. B. (1905). The development of the Leishman body in culti-vation. *Journ. R. Army Med. Corps.*, IV, 321.

LEMAIRE, G., SERGENT, E., & LHÉRITIER, A. (1913). Recherches sur la leishmaniose du chien d'Alger. *Bull. Soc. Path. Exot.*, VI, 579.

LEON, W. DE (1919). *Balantidium haughwouti*, new species, parasitic in the intestinal tract of *Ampullaria* species, a morphological study. With remarks on the relation between the mega-nucleus and the micronucleus.

LERCHE, M. (1921). Die Coccidiose der Schafe. *Arch. Protist.*, XLII, 380.

—— (1923). Nierencoccidiose bei Hausgänsen. *Zeitschr. Infektionskr. d. Haust.*, XXV, 122.

LERMANTOFF, E. (1913). Ueber *Myriospora trophoniae* n. g., n. sp., ein neues, in *Trophonia plumosa* parasitierendes Coccidium. *Arch. Protist.*, XXXII, 205.

LESTOQUARD, F. (1924). Les piroplasmoses du mouton en Algérie. (Note préliminaire.) *Bull. Soc. Path. Exot.*, XVII, 122.

—— (1924a). Deuxième note sur les piroplasmoses du mouton en Algérie. L'anaplasmose: *Anaplasma ovis* nov. sp. *Ibid.*, XVII, 784.

—— (1925). Troisième note sur les piroplasmoses du mouton en Algérie. La piroplasmose vraie: *Piroplasma* (s. str.) *ovis* (nov. sp.). Comparaison avec *Babesiella ovis*. *Ibid.*, XVIII, 140.

LEUCKART, R. (1860). *Untersuchungen über Trichina spiralis*. *Zugleich ein Beitrag zur Kenntnis der Wurmkrankheiten*, Leipzig, 11.

—— (1861). Bericht über die wissenschaftlichen Leistungen in der Naturgeschichte der niederen Thiere während des Jahres 1859. *Arch. Naturgesch.*, XXVI, ii, 103.

—— (1861a). Ueber *Paramoecium* (?) *coli* Malmst. *Ibid.*, XXVII, i, 81.

—— (1863). *Die menschlichen Parasiten*, I. Leipzig.

—— (1879). *Die Parasiten des Menschen*, 2nd edition, I. Leipzig.

—— (1886). *The parasites of man*. English translation. London.

LEVADITI, C., & MUTERMILCH, ST. (1909). Recherches sur la méthode de Bordet et Gengou appliquée à l'étude des trypanosomiases. *Zeitschr. Imm.-Forsch.*, II, 702.

—— —— (1910). Mécanisme de la phagocytose. *C. R. Soc. Biol.*, LXVIII, 1079.

—— —— (1911). Le diagnostic de la maladie du sommeil par l'examen des propriétés attachantes du sérum. *C. R. Acad. Sci.*, CLIII, 366.

LEVADITI, C., NICOLAU, S., & SCHOEN, R. (1923). L'étiologie de l'encéphalite. *Ibid.*, CLXXVII, 985.

LEVADITI, C., NICOLAU, S. & SCHOEN, R. (1924). Nouvelles recherches sur l'*Encephalitozoon cuniculi*. *C. R. Soc. Biol.*, XC, 662.

—— (1924a). L'étiologie de l'encéphalite épizootique du lapin, dans ses rapports avec l'étude expérimentale de l'encéphalite léthargique. *Encephalitozoon cuniculi* (nov. spec.). *Ann. Inst. Pasteur*, XXXVIII, 651.

—— —— —— (1924b). La nature du virus rabique. *C. R. Soc. Biol.*, XC, 994.

—— —— —— (1924c). La nature microsporidienne du virus rabique. *Ibid.*, XC, 398.

LEWIN, K. R. (1913). The nuclear structure and sporulation of *Agrippina bona* Strickland. *Parasitology*, VI, 257.

LEWIS, T. R. (1870). A report on the microscopic objects found in cholera evacuations, etc. Appendix in *Ann. Rep. San. Commissioner Govt. India*, Calcutta, 1869, No. VI, 126.

—— (1878). The microscopic organisms found in the blood of man and animals, and their relation to disease. *Ann. Rep. San. Commissioner Govt. India*, Calcutta, 1877, No. XIV, 157.

—— (1879). Flagellated organisms in the blood of healthy rats. *Quart. Journ. Micro. Sci.*, XIX, 109.

LEYDEN, E. VON, & SCHAUDINN, F. (1896). *Leydenia gemmipara* Schaudinn, ein neuer in der Ascitesflüssigkeit des lebenden Menschen gefundener, amöbenähnlicher Rhizopode. *Sitzungsb. Akad. Wiss. Berlin*, XXIX, 951.

LEYDIG, F. (1857). *Lehrbuch der Histologie des Menschen und der Thiere*, Frankfurt, 346.

LIBERT, E. (1924). Le tubage duodénal. (Thèse.) Review in *Bull. Inst. Pasteur*, XXII, 305.

LIBERT, E., & LAVIER, G. (1923). Sur l'habitat de *Giardia intestinalis* chez l'homme. Précisions fournies par le tubage duodénal. *Bull. Soc. Path. Exot.*, XVI, 589.

LICHTENHELD, G. (1911). Beurteilung eines Befundes von Koch'schen Plasmakugeln in Niereninfarkten einer Elenantilope. *Zeitschr. Infektionskr. d. Haust.*, IX, 154.

LICHTENSTEIN, J. L. (1921). *Ophryoglena collini* n.sp., parasite coelomique des larves d'Ephémères. *C. R. Soc. Biol.*, LXXXV, 794.

LIEBERKÜHN, N. (1854). Über die Psorospermien. *Arch. Anat. & Phys.*, 1.

—— (1855). Evolution des Grégarines. *Mém. couronnés Acad. R. Belg.*, XXVI.

—— (1870). *Ueber Bewegungserscheinungen der Zellen*. Marburg.

LIEBETANZ, E. (1905). Die parasitischen Protozoen des Wiederkäuermagens. *Berlin. Tierärztl. Woch.*, XXI, 313.

—— (1910). Die parasitischen Protozoen des Wiederkäuermagens. *Arch. Protist.*, XIX, 19.

LIGNIÈRES, J. (1901). Sur la "Tristeza." *Ann. Inst. Pasteur*, XV, 121.

—— (1903). La piroplasmose bovine. Nouvelles recherches et observations sur la multiplicité des parasites, leur évolution, la transmission naturelle de la maladie et la vaccination. *Arch. Parasit.*, VII, 398.

—— (1919). Piroplasmes, anaplasmes et grains chromatiques. *Bull. Soc. Path. Exot.*, XII, 558.

—— (1924). Démonstration en France de la réceptivité des ovins à l'*Anaplasma argentinum*, par injection à des bovidés, du sang d'un mouton apporté de Buenos-Ayres. *Ibid.*, XVII, 642.

—— (1924a). Nouvelles preuves de l'inoculation de l'*Anaplasma* du boeuf au mouton, puis du mouton au mouton. Comparaison des résultats obtenus avec ceux publiés par l'Institut Pasteur d'Algérie. *Ibid.*, XVII, 647.

LIGNOS, A. (1913). L'infection par Leishmania des chiens de l'île d'Hydra. *Ibid.*, VI, 117.

—— (1916). La leishmaniose canine à Hydra. *Ibid.*, IX, 302.

LINDEMANN, K. (1865). Weiteres über Gregarinen. *Bull. Soc. Imp. Nat. Moscou*, XXXVIII, ii, 381.

LINDENBERG, A. (1909). L'ulcère de Bauru ou le bouton d'orient au Brésil. *Bull. Soc. Path. Exot.*, II, 252.

LINGARD, A. (1893, 1898). Report on horse surra—I, II.

—— (1899). Report on surra in equines, bovines, buffaloes, and canines, together with an account of experiments conducted with the trypanosoma of rats, bandicoots, and fish—II. Bombay.

—— (1903). The giant *Trypanosoma* discovered in the blood of bovines. *Centralbl. Bakt.*, I. Abt., XXXV, 234.

—— (1904). A short account of the various *Trypanosomata* found to date in the blood of some of the lower animals and fish. *Indian Med. Gaz.*, XXXIX, 445.

—— (1907). Different species of *Trypanosomata* observed in bovines in India. *Journ. Trop. Vet. Sci.*, II, 4.

LINGARD, A., & JENNINGS, E. (1904). A preliminary note on a piroplasmosis found in man and in some of the lower animals. *Indian Med. Gaz.*, XXXIX, 161.

LISTER, J. J. (1895). Contributions to the life-history of the Foraminifera. *Phil. Trans. Roy. Soc.* (B), CLXXXVI, 401.

LISTON, W. G., & MARTIN, C. H. (1911). Contributions to the study of pathogenic amoebae from Bombay. *Quart. Journ. Micro. Sci.*, LVII, 107.

LITTEN (1886). Ueber Hydropneumothorax und das Auftreten von Cercomonaden im lebenden Lungengewebe. *Verhandl. V. Congr. Inn. Med.*, Wiesb., 417.

LLOYD, LL. (1912). Notes on *Glossina morsitans* in Northern Rhodesia. *Bull. Entom. Res.*, III, 95.

—— (1924). Note on the occurrence of a Herpetomonad in *Glossina morsitans*. *Ibid.*, XV, 185.

LLOYD, LL., & JOHNSON, W. B. (1924). The trypanosome infections of tsetse flies in Northern Nigeria and a new method of estimation. *Ibid.*, XIV, 265.

LLOYD, LL., JOHNSON, W. B., YOUNG, W. A., & MORRISON, H. (1924). Second report of the Tsetse-fly Investigation in the Northern Provinces of Nigeria. *Ibid.*, XV, 1.

LOON, F. H. VAN, & KIRSCHNER, L. (1924). Inoculation of benign tertian malaria from man on man in Batavia. *Meded. Burgerlijk. Geneesk. Dienst Ned.-Indië* (foreign edition), No. 3, 271.

LÖSCH, F. (1875). Massenhafte Entwickelung von Amöben im Dickdarm. *Arch. Path. Anat.*, LXV, 196.

LOUNSBURY, C. P. (1901). Transmission of malignant jaundice of the dog by a species of tick. *Agric. Journ. Cape of Good Hope.* XIX, 714.

—— (1902). Ticks and African coast fever. *Rept. Govt. Entomol. Cape of Good Hope*, 16.

—— (1903). Ticks and African coast fever. *Ibid.*, 11.

—— (1904). Transmission of African coast fever. *Agric. Journ. Cape of Good Hope*, XXIV, 428.

—— (1904a). Ticks and malignant jaundice of the dog. *Journ. Comp. Path. & Therap.*, XVII, 113.

—— (1906). Ticks and African coast fever. *Agric. Journ. Cape of Good Hope*, XXVIII, 634.

LOW, G. C. (1920). A case of oriental sore cured by intravenous injections and inunctions of antimonium tartaratum. *Journ. State Med.*, XXVIII, 354.

—— (1920a). Some unusual forms of dysentery. *Brit. Med. Journ.*, I, 255.

LOW, G. C., & DOBELL, C. (1916). Three cases of *Entamœba histolytica* infection treated with emetine bismuth iodide. *Lancet*, II, 319.

LOW, G. C., & MANSON-BAHR, P. (1923). The treatment of human trypanosomiasis by " Bayer 205." *Trans. Roy. Soc. Trop. Med. & Hyg.*, XVI, 339.

LUCET, A. (1913). Recherches expérimentales sur la coccidiose du lapin domestique. *C. R. Acad. Sci.*, CLVII, 1091.

LÜHE, M. (1902). Über Geltung und Bedeutung der Gattungsnamen *Eimeria* und *Coccidium*. *Centralbl. Bakt.*, I. Abt., XXXI. 771.

—— (1906). Die im Blute schmarotzenden Protozoen und ihre nächsten Verwandten. Mense's *Handb. Tropenkrankh.*, III.

LUTZ, A. (1901). Ueber die Drepanidien der Schlangen. Ein Beitrag zur Kenntnis der Hämosporidien. *Centralbl. Bakt.*, I. Abt., XXIX, 390.

LUTZ, A., & SPLENDORE, A. (1903). Ueber Pebrine und verwandte Mikrosporidien. *Ibid.*, I. Abt., XXXIII, 150.

—— —— (1904). Ueber Pebrine und verwandte Microsporidien. Nachtrag zur ersten Mitteilung. *Ibid.*, I. Abt., XXXVI, 645.

—— —— (1908). Ueber Pebrine und verwandte Mikrosporidien. *Ibid.*, I. Abt., XLVI, 311.

LWOFF, A. (1924). Infection expérimentale à *Glaucoma piriformis* (infusoire) chez *Galleria mellonella* (lépidoptère). *C. R. Acad. Sci.*, CLXXVIII, 1106.

—— (1925). Chytridinées parasites des amibes de l'homme. Possibilité de leur utilisation comme moyen biologique de lutte contre la dysenterie amibienne. *Bull. Soc. Path. Exot.*, XVIII, 18.

LYNCH, K. M. (1915). The rat a carrier of a dysenteric amœba. *Journ. Amer. Med. Assoc.*, LXV, 2232.

—— (1915a). Trichomoniasis of the vagina and the mouth: cultivation of the causative organism and experimental infection. A preliminary communication. *Amer. Journ. Trop. Dis. & Prev. Med.*, II, 627.

—— (1915b). Concerning endamebiasis of the mouth. *Ibid.*, II, 231.

—— (1915c). Clinical and experimental trichomoniasis of the intestine. With cultivation of the causative organism. *New York Med. Journ.*, CI, 886.

—— (1916). Dauercyst formation of *Trichomonas intestinalis*. *Journ. Parasit.*, III, 28.

—— (1920). Penetration of the intestine by *Endamœba histolytica*. *Journ. Amer. Med. Assoc.*, LXXV, 5.

—— (1922). Cultivation of Trichomonas and the question of differentiation of the flagellates. *Ibid.*, LXXIX, 1130.

—— (1922a). *Tricercomonas intestinalis* and *Enteromonas caviæ*, n. sp., and their growth in culture. *Journ. Parasit.*, IX, 29.

—— (1924). Ingestion of red blood-corpuscles by an intestinal amœba with eight-nucleated cyst. *Amer. Journ. Trop. Med.*, IV, 43.

MAC ARTHUR, W. P. (1922). A holotrichous ciliate pathogenic to *Theobaldia annulata* Schrank. *Journ. R. Army Med. Corps*, XXXVIII, 83.

MacCALLUM, W. G. (1897). On the flagellated form of the malarial parasite. *Lancet*, II, 1240.

MacDOUGALL, M. S. (1925). Cytological observations on gymnostomatous ciliates, with a description of the maturation phenomena in diploid and tetraploid forms of *Chilodon uncinatus*. *Quart. Journ. Micro. Sci.*, LXIX, 361.

MACFIE. J. W. S. (1913). Trypanosomiasis of domestic animals in Northern Nigeria. *Ann. Trop. Med. & Parasit.*, VII, 1.

—— (1913a). On the morphology of the trypanosome (*T. nigeriense*, n. sp.) from a case of sleeping sickness from Eket, Southern Nigeria. *Ibid.*, VII, 339.

—— (1914). Trypanosomes found infecting wild *Glossina tachinoides*. *Ibid.*, VIII, 417.

—— (1914a). Notes on some blood parasites collected in Nigeria. *Ibid.*, VIII, 439.

—— (1915). Babesiasis and trypanosomiasis at Accra, Gold Coast, West Africa. *Ibid.*, IX, 457.

—— (1915a). A case of dysentery in a monkey, in which amœbæ and spirochætes were found. *Ibid.*, IX, 507.

—— (1916). The results of dissections of tsetse flies at Accra. *Rept. Accra Lab.*, 1915, London.

—— (1917). Examinations of various animals—dogs. *Ibid.*, 1916, London, 25.

—— (1917a). A parasite of mosquitoes *Lankesteria culicis* Ross, 1898. *Ibid.*, 1916. London, 73.

—— (1917b). Preliminary note on a monomorphic trypanosome found in the blood of a native of the Gold Coast. *Brit. Med. Journ.*, 1, 12.

MACFIE. J. W. S., & INGRAM, A. (1922). Accra laboratory report for 1921. *Rept. Med. Dept. Gov. Gold Coast*.

MACHADO. A. (1911). Zytologische Untersuchungen über *Trypanosoma rotatorium* Gruby. *Mem. Inst. O. Cruz*, III, 108.

—— (1911a). Nouvelle coccidiose de l'intestin d'un hémiptère. (Note préliminaire.) *Bull. Soc. Path. Exot.*, IV, 616.

—— (1911b). Sobre um novo coccidio do intestino de um hemiptero. *Brazil-Medico*, XXV, 389.

—— (1913). Citolojia e ciclo evolutivo da *Chagasella alydi*, novo coccidio parazito dum hemiptero do genero *Alydus*. (Also in German.) *Mem. Inst. O. Cruz*, V, 32.

MACKIE, F. P. (1914). Kala-azar in Nowgong (Assam). *Indian Journ. Med. Res.*, I, 626.

—— (1914a). Note on the parasite of bat malaria. *Ibid.*, II, 375.

—— (1914b). A flagellate infection of sand flies. *Ibid.*, II, 377.

—— (1914c). Note on some bodies of unknown nature found in the fæces of kala-azar patients *Ibid.*, II, 510.

—— (1915). Insects and kala-azar. *Ibid.*, II, 942.

—— (1915a). The presence of leishmania in the peripheral blood of cases of kala-azar in Assam. *Ibid.*, III, 90.

—— (1915b). The experimental transmission of Indian kala-azar to animals. *Ibid.*, II, 934.

MACKIE, F. P., GUPTA, B. M. DAS, & SWAMINATH, C. S. (1923). Progress report on kala-azar. *Ibid.*, XI, 591.

MACKINNON, D. L. (1909). Note on two new flagellate parasites in fleas—*Herpetomonas ctenophthalmi*, n. sp., and *Crithidia hystrichopsyllæ*, n. sp. *Parasitology*, II, 288.

—— (1910). Herpetomonads from the alimentary tract of certain dung flies. *Ibid.*, III, 255.

—— (1911). On some more Protozoan parasites from Trichoptera. *Ibid.*, IV, 28.

—— (1912). Protists parasitic in the larva of the crane fly, *Tipula* sp. Preliminary note. *Ibid.*, V, 175.

—— (1913). Studies on parasitic Protozoa—II. (*a*) The encystment of *Rhizomastix gracilis* Alexeieff: (*b*) *Tetratrichomastix parisii*, n. subgen., n. sp. *Quart. Journ. Micro. Sci.*, LIX, 459.

—— (1914). Observations on *Amœbæ* from the intestine of the crane-fly larva, *Tipula* sp. *Arch. Protist.*, XXXII, 267.

—— (1915). Studies on parasitic Protozoa—III. (*a*) Notes on the flagellate *Embadomonas*. (*b*) The multiplication cysts of a trichomastigine. *Quart. Journ. Micro. Sci.*, LXI, 105.

MACKINNON, D. L., & ADAM, D. 1. (1924). Notes on Sporozoa parasitic in Tubifex. I. The life-history of *Triactinomyxon* Stolc. *Ibid.*, LXVIII, 187.

MacNEAL, W. J. (1904). The life-history of *Trypanosoma lewisi* and *Trypanosoma brucei*. *Journ. Infect. Dis.*, I, 517.

MAGGIO, C., & ROSENBUSCH, F. (1915). Studien über die Chagaskrankheit in Argentinien und die Trypanosomen der "Vinchucas" (Wanzen, *Triatoma infestans* Klug). *Centralbl. Bakt.*, I. Abt., LXXVII, 40.

MAITRA, J. N. (1924). A flagellate found in the peripheral blood-smear of a case clinically one of kala-azar. *Calcutta Med. Journ.*, XVIII, 618.

MALIWA. E., & HAUS, V. VON (1920). Ueber Balantidieninfektion der Harnwege. *Zeitschr Urol.*, XIV, 495.

MALMSTEN, P. H. (1857). Infusorien als Intestinal-Tiere beim Menschen. *Arch. Path. Anat.*, XII, 302.

MANIFOLD, J. A. (1924). Report on a case of sarcosporidiosis in a human heart. *Journ. R. Army Med. Corps*, XLII, 275.

MANLEITNER (1912). Piroplasmose unter Giraffen. *Med. Berichte Deutsch. Schutzgeb.*, 1910-11, 293.

MANLOVE, C. H. (1917). Two cases of balantidial colitis. *Philippine Journ. Sci.*, (B), XII, 149.

MANOUELIAN, Y., & VIALA, J. (1924). " *Encephalitozoon rabiei* " parasite de la rage. *Ann. Inst. Pasteur*, XXXVIII, 258.

MANSON, P. (1894). On the nature and significance of the crescentic and flagellated bodies in malarial blood. *Brit. Med. Journ.*, II, 1306.

—— (1898). The mosquito and the malaria parasite. *Ibid.*, II, 849.

—— (1900). Experimental proof of the mosquito malaria theory. *Ibid.*, II, 949.

—— (1905). Notes on two cases of febrile tropical splenomegaly (kala-azar) and a suggestion. *Ibid.*, II, 1261.

MANSON, P., & LOW. G. C. (1904). The Leishman-Donovan body in ulcerated surfaces. *Ibid.*, II, 11.

MANSON, P., & SAMBON, L. W. (1909). A case of intestinal pseudo-parasitism due to *Chilodon uncinatus* (Blochmann). *Lancet*, I, 832.

MANTOVANI, M. (1912). Infezione sperimentale da *Leishmania donovani* nel coniglio. *Pathologica*, IV, 415.

MARCHAND, F. (1875). Ein Fall von Infusorien im Typhus-Stuhl. *Arch. Path. Anat.*, LXIV, 293.

—— (1894). Ueber das Vorkommen von *Trichomonas* im Harne eines Mannes, nebst Bemerkungen über *Trichomonas vaginalis*. *Centralbl. Bakt.*, I. Abt., XV. 709.

MARCHAND (1904). Über neue Protozoeninfektionen beim Menschen. *Münch. Med. Woch.*, LI, 630.

MARCHIAFAVA, E., & CELLI, A. (1889). Sulle febbri malariche predominanti nell' estate e nell' autunno in Roma. Nota preventiva. *Riforma Med.*, Naples, No. 214.

MARCHOUX, E. (1899). Note sur la dysenterie des pays chauds. *C. R. Soc. Biol.*, LI, 870.

—— (1900). *Piroplasma canis* (Laveran) chez les chiens du Sénégal. *Ibid.*, LII, 97.

MARCHOUX, E., SALIMBENI, A., & SIMOND, P. L. (1903). La fièvre jaune. Rapport de la Mission française. *Ann. Inst. Pasteur*, XVII, 665.

MARKOFF, W. N. (1916). Piroplasmose und andere blutparasitäre Krankheiten der Haustiere am Balkan. *Arch. Schiffs.- u. Trop.-Hyg.*, XX, 313.

MAROTEL, G. (1905). La coccidiose de la chèvre et son parasite. *Bull. Soc. Sci. Vét. Lyon*, 52.

—— (1906). Coccidiose et coccidies chez la chèvre. *Bull. Soc. Centr. Méd. Vét.*, LX, 373.

—— (1921). Sur une nouvelle coccidie du chat. *Bull. Soc. Sci. Vét. Lyon*, 86.

MARSHALL, D. G. (1912). A case of amœbic dysentery in a man who had never been out of Scotland. *Edinburgh Med. Journ.*, VIII, 229.

MARSHALL, W. E. (1911). Experimental kala-azar in the grey monkey of the Sudan (*Cercopithecus sabæus*). *Journ. R. Army Med. Corps*, XVII, 255.

—— (1911a). Kala-azar commission to investigate the prevalence and cause of the disease in the Eastern Sudan. (2) Pathological report. *4th Rept., Wellcome Trop. Res. Labs., Khartoum*, A, 157.

—— (1912). Further experimental investigation into Sudan kala-azar. *Journ. R. Army Med. Corps*, XIX, 276.

MARTIN, C. H. (1910). Observations on *Trypanoplasma congeri*. *Quart. Journ. Micro. Sci.*, LV, 485.

—— (1912). A note on the Protozoa from sick soils, with some account of the life-cycle of a flagellate Monad. *Proc. Roy. Soc.*, B, LXXXV, 393.

—— (1913). Further observations on the intestinal trypanoplasmas of fishes, with a note on the division of *Trypanoplasma cyprini* in the crop of a leech. *Quart. Journ. Micro. Sci.*, LIX, 175.

MARTIN, C. H., & LEWIN, K. R. (1914). Some notes on soil Protozoa. *Phil. Trans. Roy. Soc.*, B, CCV, 77.

MARTIN, C. H., & ROBERTSON, M. (1911). Further observations on the cæcal parasites of fowls, with some reference to the rectal fauna of other vertebrates. Part I. *Quart. Journ. Micro. Sci.*, LVII, 53.

MARTIN, C. J., KELLAWAY, & WILLIAMS, F. E. (1918). Epitome of the results of the examination of the stools of 422 cases admitted to No. 3 Australian General Hospital, Cairo, for dysentery and diarrhœa, March to August, 1916. *Journ. R. Army Med. Corps*, XXX, 101.

MARTIN, F. P. (1907). Über Darmcoccidiose bei der Hausziege. *Berlin. Tierärztl. Woch.*, XXIII, 6.

MARTIN, G. (1907). Sur un trypanosome de saurien (*Trypan. boueti*, n. sp.). *C. R. Soc. Biol.*, LXII, 594.

MARTIN, G., LEBŒUF, & ROUBAUD (1909). Trypanosomes d'animaux divers au Moyen-Congo. *Bull. Soc. Path. Exot.*, II, 209.

MARTIN, L., & DARRÉ, H. (1912). Un cas de trypanosomiase humaine contractée au laboratoire. *Ibid.*, V, 883.

MARTINEZ, F. F. (1915). Las leishmaniosis patogenas en el mediodía de España. *Rev. Med. y Cir. Pract.; Clin. Mod. Zaragoza*, XIV, 149, 189. Paris.

MARTINEZ, I. G. (1914). Canine babesiasis in Porto Rico. *Journ. Trop. Med. & Hyg.*, XVII, 194.

MARTINI, E. (1910). Ueber einen bei amöbenruhrähnlichen Dysenterien vorkommenden Ciliaten. *Zeitschr. Hyg. u. Infekt.*, LXVII, 387.

—— (1909). The development of a *Piroplasma* and *Trypanosoma* of cattle in artificial culture media. *Philippine Journ. Sci.*, (B), IV, 149.

—— (1920). Ueber Stechmücken besonders deren europäische Arten und ihre Bekämpfung. *Arch. Schiffs.- u. Trop.-Hyg.*, XXIV (Beih. 1), 1.

MARTIRANO. F. (1902). La malaria nel mezzogiorno d'Italia. *Atti Soc. Studi d. Malaria*, Rome, III, 475.

MARTOGLIO, F. (1911). La peste bovina e le tripanosomiasi nella Somalia italiana. *Annali d'Igiene*, XXI, 453.

—— (1913). Contributo alla conoscenza delle leucocytogregarine. *Ibid.*, XXIII, 161.

—— (1917). Su di un nuovo genere di parassiti del sangue. *Ibid.*, XXVII, 561.

MARTOGLIO, F., & CARPANO, M. (1906). Sopra un caso di *Hæmogregarina bovis*. *Ibid.*, XVI, 251.

MARTOGLIO, F., STELLA, V., & CARPANO, M. (1910). Su un plasmodio della scimia. *Ibid.*, XX, 287.

MARULLAZ, M. (1913). Au sujet d'un toxoplasme des oiseaux. *Bull. Soc. Path. Exot.*, VI, 323.

—— (1920). Sur l'évolution de *Sarcocystis muris*. *Ann. Inst. Pasteur*, XXXIV, 547.

MARZINOWSKY, E. J. (1909). Cultures de *Leishmania tropica* (s. *Ovoplasma orientale*, s. *Helcosoma tropicum*) parasite du bouton d'orient. *Bull. Soc. Path. Exot.*, II, 591.

—— (1916). De différentes espèces du parasite de la malaria. *Ann. Inst. Pasteur*, XXX, 243.

—— (1917). [On *Grahamella*.] *Medicinsk. Obozrenije*, Moscow, Nos. 1-2. (In Russian.)

MARZINOWSKY, E. J., & BIELITZER, A. W. (1909). Piroplasmose des Pferdes in Russland und die Rolle der Zecke *Dermacentor reticulatus* bei ihrer Verbreitung. *Zeitschr. Hyg. u. Infekt.*. LXIII, 17.

MARZINOWSKY, E. J., & BOGROFF, S. L. (1904). Etiologie du bouton d'orient. *Medicinsk. Obozrenije*, Moscow, LXI, 207. (In Russian.)

MARZOCCHI, V. (1911). Di un flagellato parassita del tubo digerente del *Ctenocephalus canis* L. *Pathologica*, III, 256.

MASON, F. E. (1915). A new form of Piroplasmosis in Sudanese sheep. *Min. Agric., Vet. Serv., Ann. Rept.*, 1914, Cairo, 29.

—— (1916). Annual report for the year 1915 of the Veterinary Pathological Laboratory *Ibid.*, 1915.

—— (1922). Egyptian fever in cattle and buffaloes. *Journ. Comp. Path. & Therap.*, XXXV, 33.

MATHIS. C. (1906). Sur une modification au milieu de Novy-MacNeal pour la culture des trypanosomes. *C. R. Soc. Biol.*, LXI, 550.

—— (1909). La piroplasmose canine au Tonkin. *Bull. Soc. Path. Exot.*, II, 380.

—— (1913). Entamibes des singes. *Bull. Soc. Méd.-Chir. Indochine*, IV, 388.

—— (1914). Evolution d'un trypanosome dans le liquide salivaire d'un moustique. *C. R. Soc. Biol.*, LXXVII, 297.

MATHIS, C., & LEGER, M. (1909). Présence d'un leucocytozoaire chez les chiens du Tonkin. *Ibid.*, LXVII, 98.

—— —— (1909a). Sur un nouveau trypanosome des serpents du Tonkin. *Ibid.*, LXVII, 572.

—— —— (1911). *Plasmodium* des macaques du Tonkin. *Ann. Inst. Pasteur*, XXV, 593.

—— —— (1911a). *Recherches de parasitologie et de pathologie humaines et animales au Tonkin.* Paris.

MATHIS. C., & MERCIER, L. (1917). La schizogonie chez les entamibes intestinales de l'homme. *Bull. Soc. Path. Exot.*, X, 311.

MATTHEWS, J. R. (1919). A mensurative study of the cysts of *Entamœba coli*. *Ann. Trop. Med. & Parasit.*, XII, 259.

MAUPAS, E. (1881). Contribution à l'étude des Acinétiens. *Arch. Zool. Exp.*, IX, 299.

—— (1888). Recherches expérimentales sur la multiplication des infusoires ciliés. *Ibid.*, VI, 165.

—— (1889). Le rajeunissement karyogamique chez les ciliés. *Ibid.*, VII, 149.

MAURER, G. (1902). Die Malaria perniciosa. *Centralbl. Bakt.*, I. Abt., XXXII, 695.

MAVOR, J. W. (1916). On the life-history of *Ceratomyxa acadiensis*, a new species of Myxosporidia from the eastern coast of Canada. *Proc. Amer. Acad. Arts & Sci.*, LI, 551.

MAXCY, K. F. (1921). *Giardia (Lamblia) intestinalis.* A common Protozoan parasite of children. *Johns Hopkins Hosp. Bull.*, XXXII, 166.

MAXWELL, J. L. (1912). Fistulous diseases of the buttocks: a clinical entity. *Trans. Soc. Trop. Med. & Hyg.*, VI, 50.

MAYA, E. (1912). *Annual report on the Bacteriological Laboratory for 1911.* Colony of Mauritius.

MAYER, C. (1843). *Specilegium observationum anatomicarum de organo electrico in Raiis anelectricis et de hæmatozois.* Bonn.

MAYER, M. (1908). Über Malariaparasiten bei Affen. *Arch. Protist.*, XII, 314.

MAYER, M. (1918). Über den Dauerparasitismus von *Schizotrypanum cruzi* bei *Ornithodorus moubata*. *Arch. Schiffs.- u. Trop.-Hyg.*, XXII, 158.

—— (1919). Klinische, morphologische und experimentelle Beobachtungen bei Amöbenerkrankungen. *Ibid.*, XXII, 158.

—— (1920). Über die Wanderung der Malariasichelkeime in den Stechmücken und die Möglichkeit ihrer Überwinterung in diesen. *Med. Klin.*, XVI, 1290. (Summary in *Arch. Schiffs.- u. Trop.-Hyg.*, 1921, XXV. 64.)

—— (1921). Über einige bakterienähnliche Parasiten der Erythrozyten bei Menschen und Tieren. *Arch. Schiffs.- u. Trop.-Hyg.*, XXV, 150.

MAYER, M., BORCHARDT, W., & KIKUTH. W. (1926). Über Einschlüsse der Erythrozyten bei experimenteller Anämic der Ratten (eine neue Parasitengruppe ?) *Klin. Woch.*, V. 559).

MAYER. M., & DA ROCHA-LIMA, H. (1914). Zum Verhalten von *Schizotrypanum cruzi* in Warmblütern und Arthropoden. *Arch. Schiffs.- u. Trop.-Hyg.*, XVIII (Beih. 5), 257.

MAYER, M., & WERNER, H. (1914). Kultur des Kala-Azar-Erregers (*Leishmania donovani*) aus dem peripherischen Blut des Menschen. *Deutsch. Med. Woch.*, XL, 67.

MAYER. M., & ZEISS. H. (1920). Versuche mit einem neuen Trypanosomenheilmittel (" Bayer 205 ") bei menschen- und tierpathogenen Trypanosomen. *Arch. Schiffs.- u. Trop.-Hyg.*, XXIV. 257.

MAYNARD, G. D. (1915). The trypanosomes of sleeping sickness, being a study of the grounds for the alleged identity of *T. brucei* with those causing disease in man in Nyasaland. *Pub. S. African Inst. Med. Res.*. No. VI.

MAYNE, B. (1920). Can the mosquito convey infection from a malarial patient undergoing treatment ? Does sporogony affect mosquito life ? *U.S. Publ. Health Rep.*. XXXV, 1664.

—— (1922). How long does a mosquito retain malaria parasites ? *Ibid.*, XXXVII, 1059.

MAZZANTI (1900). Enterite psorospermica da *Coccidium oviforme* Leuck. nell' agnello. *Vet. di Campagna*. IV, vii.

McCARRISON, R. (1919). The pathogenesis of deficiency disease. *Indian Journ. Med. Res.*, VII, 283.

McCULLOCH. I. (1915). An outline of the morphology and life-history of *Crithidia leptocoridis*, sp. nov. *Univ. California Publ. Zool.*, XVI, 1.

—— (1917). *Crithidia euryophthalmi*, sp. nov., from the hemipteran bug, *Euryophthalmus convivus* Stål. *Ibid.*, XVIII. 75.

—— (1919). A comparison of the life-cycle of *Crithidia* with that of *Trypanosoma* in the invertebrate host. *Ibid.*, XIX. 135.

McDONALD, J. D. (1922). On *Balantidium coli* (Malmsten) and *Balantidium suis* (sp. nov.), with an account of their neuromotor apparatus. *Ibid.*, XX. 243.

McGILL. C. (1922). Giardiasis. *Journ. Amer. Med. Assoc.*. LXXVIII, 179.

McLAY. K. (1922). Hematological investigations on malaria in Macedonia. *Journ. R. Army Med. Corps*, XXXVIII, 93.

MECKEL VON HEMSBACH. J. H. (1847). Ueber schwarzes Pigment in der Milz und dem Blute einer Geisteskranken. *Zeitschr. Psychiatr.*. 198.

MELENEY, H. E. (1925). The histopathology of kala-azar in the hamster, monkey, and man. *Amer. Journ. Path.*, I, 147.

MELLO, F. DE (1915). Preliminary note on a new hæmogregarine found in the pigeon's blood. *Indian Journ. Med. Res.*, III. 93.

—— (1916). Quelques considérations sur les affinités zoologiques du genre *Hæmocystidium*. avec description d'une espèce nouvelle. *An. Sci. Fac. Med. Porto*, III. 1.

—— (1921). Protozoaires parasites du " *Pachelabra mœsta* " Reeve. *C. R. Soc. Biol.*, LXXXIV. 241.

MELLO. F. DE, & BRAZ DE SA. L. J. (1916). A contribution to the study of Hæmoprotozoa in Portuguese India. *Indian Journ. Med. Res.*, III. 731.

MELLO, F. DE, & PAES, S. (1923). Sur une plasmodie du sang des chèvres. *C. R. Soc. Biol.*, LXXXVIII, 829.

MELLO, F. DE, & SUCTANCAR, C. (1922). Morphologie et cycle évolutif d'un *Herpetomonas* de l'intestin d'*Hemidactylus brookei* Gray. *Bull. Soc. Path. Exot.*, XV, 795.

MELLO, U. (1910). Un cas de toxoplasmose du chien, observé à Turin. *Ibid.*, III, 359.

—— (1923). L'amebiasi nei primati. *Annali d'Igiene*, XXXII, 533.

MENDELEEFF-GOLDBERG, P. (1913). Die Immunitätsfrage bei der Trypanosomenkrankheit der Frösche. *Arch. Protist.*, XXXI. 241.

MERCIER. L. (1910). Contribution à l'étude de l'amibe de la blatte (*Entamœba blattœ* Bütschli). *Ibid.*, XX, 143.

MERCIER, L., & MATHIS, C. (1918). Kystes gamogoniques et schizogoniques chez *Entamœba ranarun*. *Bull. Soc. Path. Exot.*, XI, 47.

MERESCHKOWSKY, C. VON (1879). Studien über Protozoen des nördlichen Russland. *Arch. Mikr. Anat.*, XVI, 153.

MESNIL, F. (1899). Essai sur la classification et l'origine des sporozoaires. *Cinquanten. d. l. Soc. Biol.*, vol. jubil., 258.

MESNIL, F. (1900). Sur la conservation du nom générique *Eimeria* et la classification des coccidies. *C. R. Soc. Biol.*, LII, 603.
—— (1903). Les travaux récents sur les coccidies. *Bull. Inst. Pasteur*, I, 473, 505.
—— (1907). Sur une coccidie de la rainette. *C. R. Assoc. Franç. Avanc. Sci.*, XXXVI, i, 253.
—— (1910). Sur l'identification de quelques trypanosomes pathogènes. *Bull. Soc. Path. Exot.*, III. 376.
—— (1912). [On paper by Brimont, E. (1912).] *C. R. Soc. Biol.*, LXXII, 884.
—— (1914). Pentatrichomonas. *Bull. Inst. Pasteur*, XII. 968.
—— (1916). [On paper by Weidman (1915) on *Coccidium bigeminum*.] *Ibid.*, XIV. 130.
—— (1918). [On paper by França (1918) on the classification of Hæmatozoa.] *Ibid.*, XVI, 536.
—— (1919). [On paper by du Toit, P. J. (1918).] *Ibid.*, XVII, 193.
—— (1919a). [On paper by Dobell, C. (1919), on Coccidia parasitic in man.] *Ibid.*, XVII. 376.
—— (1921). La "flagellose" ou "leptomoniase" des euphorbes et des asclépiadacées. *Ann. Sci. Nat.* (Bot.), III, Act. biol.. xlii.
MESNIL, F., & BLANCHARD, M. (1914). Sur l'identification du virus d'un cas de trypanosomiase humaine contractée au laboratoire. *Bull. Soc. Path. Exot.*, VII, 196.
—————— (1916). Sensibilité au sérum humain normal de trypanosomes d'origine humaine. *Ibid.*, IX. 81.
MESNIL, F., & BRIMONT, E.' (1908). Sur les propriétés préventives du sérum des animaux trypanosomiés. *C. R. Soc. Biol.*, LXV, 77.
—————— (1908a). Sur un hématozoaire nouveau (*Endotrypanum* n. gen.) d'un édenté de Guyane. *Ibid.*, LXV. 581.
—————— (1908b). Sur les propriétés des races de trypanosomes résistantes aux médicaments. *Ann. Inst. Pasteur*, XXII. 856.
—————— (1909). Sur les propriétés protectrices du sérum des animaux trypanosomiés. Races résistantes à ces sérums. *Ibid.*, XXIII. 129.
—————— (1910). Trypanosome et microfilaire d'un édenté, le *Tamandua tridactyla* (L.). *C. R. Soc. Biol.*, LXIX. 148.
MESNIL, F., & MARTIN, G. (1906). Sur la réceptivité des oiseaux aux trypanosomes pathogènes pour les mammifères. *Ibid.*, LX, 739.
MESNIL, F., & NICOLLE, M. (1906). Traitement des trypanosomiases par les couleurs de benzidine. 2e partie: étude expérimentale. *Ann. Inst. Pasteur*, XX, 513.
MESNIL, F., & RINGENBACH, J. (1911). De l'action des sérums de primates sur le trypanosome humain de Rhodesia. *C. R. Acad. Sci.*, CLIII, 1097.
—————— (1911a). Sur les affinités du trypanosome humain de Rhodesia et du *T. gambiense*. *C. R. Soc. Biol.*, LXXI. 271.
—————— (1911b). Action pathogène du *Trypanosoma rhodesiense*. *Bull. Soc. Path. Exot.*, IV, 675.
—————— (1912). De l'action des sérums de primates sur les trypanosomes humains d'Afrique. *C. R. Acad. Sci.*, CLV, 78.
—————— (1912a). Observation d'une chèvre infectée de *Trypanosoma rhodesiense*. *Bull. Soc. Path. Exot.*, V. 105.
—————— (1912b). Au sujet de la comparaison des *Trypanosoma gambiense* et *rhodesiense*. *C. R. Soc. Biol.*, LXXII, 58.
MESNIL, F., & ROUBAUD, E. (1917). Sur la sensibilité du chimpanzé au paludisme humain. *C. R. Acad. Sci.*, CLXV, 39.
—————— (1920). Essais d'inoculation du paludisme au chimpanzé. *Ann. Inst. Pasteur*, XXXIV, 466.
METCALF, M. M. (1907). Studies on *Opalina*. (Preliminary notice.) *Zool. Anz.*, XXXII, 110.
—— (1909). *Opalina*. Its anatomy and reproduction, with a description of infection experiments and a chronological review of the literature. *Arch. Protist.*, XIII, 195.
—— (1918). *Opalina* and the origin of the ciliate Infusoria. *Journ. Washington Acad. Sci.*, VIII, 427.
—— (1920). The classification of the Opalinidæ. *Science*, LII, 135.
—— (1923). The opalinid ciliate infusorians. *Smithsonian Inst., Nation. Mus.*, Bull. 120.
METZNER, R. (1901). Untersuchungen an *Megastoma entericum* Grassi aus dem Kanichendarm. *Zeitschr. Wiss. Zool.*, LXX, 299.
—— (1903). Untersuchungen an *Coccidium cuniculi*. I. Teil. *Arch. Protist.*, II, 13.
MEYER, K. F. (1909). Preliminary note on the transmission of East Coast fever to cattle by intraperitoneal inoculation of the spleen or portions of the spleen of a sick animal. *Journ. Comp. Path. & Therap.*, XXII, 213.
MEZINCESCU, D. (1909). Evolution des ookinètes d'*Hæmoproteus* dans l'intestin des moustiques. *C. R. Soc. Biol.*, LXVI, 329.
M'FADYEAN, J. (1896). Intestinal psorospermosis in lambs. *Journ. Comp. Path. & Therap.*, IX, 31.
M'FADYEAN, J., & STOCKMAN, S. (1911). A new species of piroplasm found in the blood of British cattle. *Ibid.*, XXIV, 340.

MIDDLETON, A. R. (1915). Heritable variations and the results of selection in the fission rate of *Stylonychia pustulata*. *Journ. Exp. Zool.*, XIX, 451.

MIESCHER, F. (1843). Ueber eigenthümliche Schläuche in den Muskeln einer Hausmaus. *Ber. u. d. Verhandl. Naturf. Ges. Basel*, V, 198.

MIGONE, L. E. (1910). Le rôle des carpinchos comme reservoir de virus dans la conservation du mal de Caderas. *Bull. Soc. Path. Exot.*, III, 524.

—— (1916). Un nouveau flagellé des plantes, *Leptomonas elmassiani*. *Ibid.*, IX, 356.

—— (1916a). Parasitologie de certains animaux du Paraguay. *Ibid.*, IX, 359.

—— (1917). Un nuevo flagelado de las plantas (*Leptomonas elmassiani*). *Proc. 2nd Pan-Amer. Sci. Congr.*, X, 572.

MILLER, W. W. (1908). *Hepatozoon perniciosum* (n. g., n. sp.), a hæmogregarine pathogenic for white rats, with a description of the sexual cycle in the intermediate host, a mite (*Lelaps echidninus*). Bull. No. 46, Hyg. Lab., Washington.

MINCHIN, E. A. (1907). On the occurrence of encystation in *Trypanosoma grayi* Novy, with some remarks on the method of infection in trypanosomes generally. *Proc. Roy. Soc.*, B, LXXIX, 35.

—— (1908). Investigations on the development of trypanosomes in the tsetse flies and other Diptera. *Quart. Journ. Micro. Sci.*, LII, 159.

—— (1909). Observations on the flagellates parasitic in the blood of fresh-water fishes. *Proc. Zool. Soc. London*, I, 2.

—— (1909a). The structure of *Trypanosoma lewisi* in relation to microscopical technique. *Quart. Journ. Micro. Sci.*, LIII, 755.

—— (1910). Report on a collection of blood parasites made by the Sleeping Sickness Commission, 1908-09, in Uganda. *Rep. Sleep. Sickn. Comm. Roy. Soc.*, X, 73.

—— (1910a). On some parasites observed in the rat flea (*Ceratophyllus fasciatus*). *Festschr. f. R. Hertwig*, Jena, I, 291.

—— (1912). *An introduction to the study of the Protozoa*. London.

—— (1914). Remarks on the nature of the blepharoplasts or basal granules of flagella. *Arch. Protist.*, XXXIV, 212.

—— (1914a). The development of trypanosomes in the invertebrate host. *Nature*, XCIV, 406; *Rept. Brit. Assoc.* (1914), 1915, 404.

MINCHIN, E. A., & FANTHAM, H. B. (1905). "*Rhinosporidium kenealyi*" n. g., n. sp., a new sporozoön from the mucous membrane of the septum nasi of man. *Quart. Journ. Micro. Sci.*, XLIX, 521.

MINCHIN, E. A., GRAY, A. C. H., & TULLOCH, F. M. G. (1906). *Glossina palpalis* in its relation to *Trypanosoma gambiense* and other trypanosomes. (Preliminary report.) *Proc. Roy. Soc.*, B, LXXVIII, 242.

MINCHIN, E. A., & THOMSON, J. D. (1910). The transmission of *Trypanosoma lewisi* by the rat flea (*Ceratophyllus fasciatus*). (Preliminary communication.) *Ibid.*, LXXXII, 273.

—— —— (1911). On the occurrence of an intracellular stage in the development of *Trypanosoma lewisi* in the rat flea. *Brit. Med. Journ.*, II, 361.

—— —— (1911a). The transmission of *Trypanosoma lewisi* by the rat flea (*Ceratophyllus fasciatus*). *Ibid.*, I, 1309.

—— —— (1915). The rat trypanosome, *Trypanosoma lewisi*, in its relation to the rat flea, *Ceratophyllus fasciatus*. *Quart. Journ. Micro. Sci.*, LX, 463.

MINCHIN, E. A., & WOODCOCK, H. M. (1910). Observations on certain blood parasites of fishes occurring at Rovigno. *Ibid.*, LV, 113.

—— —— (1911). Observations on the trypanosome of the little owl (*Athene noctua*), with remarks on the other Protozoan blood parasites occurring in this bird. *Ibid.*, LVII, 141.

MITZMAIN, M. B. (1913). The mechanical transmission of surra by *Tabanus striatus*, Fabricus. *Philippine Journ. Sci.*, VIII, B, 223.

—— (1915). *Anopheles* as a winter carrier of *Plasmodium*. The mosquito as a prophylactic indicator. *U.S. Publ. Health Rep.*, XXX, 2117.

—— (1916). *Anopheles punctipennis* Say: its relation to the transmission of malaria. Report of experimental data relative to subtertian malarial fever. *Ibid.*, XXXI, 301.

—— (1916a). Anopheles infectivity experiments. An attempt to determine the number of persons one mosquito can infect with malaria. *Ibid.*, XXXI, 2325.

—— (1917). The malaria parasite in the mosquito. The effects of low temperature and other factors on its development. *Ibid.*, XXXII, 1400.

—— (1917a). *Anopheles punctipennis*. A note on its ability to serve as a host for *Plasmodium falciparum*. *Ibid.*, XXXII, 1081.

MIURA, K. (1894). *Trichomonas vaginalis* in frischgelassenen Urin eines Mannes. *Centralbl. Bakt.*, I. Abt., XVI, 67.

MIYAJIMA, M. (1907). On the cultivation of a bovine *Piroplasma* : a preliminary communication. *Philippine Journ. Sci.*, II, B, 83.

MÖBIUS, K. (1888). Bruchstücke einer Infusorienfauna der Kieler Bucht. *Arch. Naturgesch.*, LIV, i, 81.

Möller, J. (1923). Kokzidien bei den Säugetieren des zoologischen Gartens zu Berlin. (Inaug. Diss. Tierärztl. Hochsch. Berlin.)

Moldovan, J. (1914). Untersuchungen über den Zeugungskreis des *Leucocytozoon ziemanni* (Laveran). *Arch. Protist.*, XXXIV, 249.

Molisch, H. (1903). Amoeben als Parasiten in *Volvox*. *Ber. Deutsch. Bot. Ges.*, XXI, 20.

Monge, C. M. (1914). La leishmaniosis del dermis en el Peru. Espundia—Uta—Juccuya—Qcepo—Tiacc-Araña. *Cronica Medica*, XXXI, 231, 251, 288, 385.

Montgomery, R. E. (1913). On bodies resembling Koch's bodies in the spleen of a topi (*Damaliscus corrigum jimela*). *Ann. Rept. Vet. Path. Lab. Nairobi, 1911-12*, 25.

Montgomery, R. E., & Kinghorn, A. (1908). A report on trypanosomiasis of domestic stock in North-Western Rhodesia. *Ann. Trop. Med. & Parasit.*, II, 97.

—— —— (1909). Concerning *Trypanosoma dimorphon* Dutton and Todd: *T. confusum* sp. nova. *Lancet*, II, 927.

Moore, E. (1923). *Octomitus salmonis*, a new species of intestinal parasite in trout. *Trans. Amer. Fish. Soc.*, LII (1922), 74.

Moore, J. E. Salvin-, & Breinl, A. (1907). The cytology of the trypanosomes. *Ann. Trop. Med. & Parasit.*, I, 441.

—— (1908). The life-history of *Trypanosoma equiperdum*. *Proc. Roy. Soc.*, B, LXXX, 288.

Moore, J. E. Salvin-, Breinl, A., & Hindle, E. (1908). The life-history of *Trypanosoma lewisi*. *Ann. Trop. Med. & Parasit.*, II, 197.

Moquin-Tandon, A. (1860). *Eléments de zoologie médicale*. Paris.

Morax. V. (1906). Les affections oculaires dans les trypanosomiases. *Ann. d'Ocul.*, CXXXVI, 437.

—— (1907). Manifestations oculaires au cours des trypanosomiases. *Ann. Inst. Pasteur*, XXI, 47.

Morgenroth, J. (1918). Die Therapie der Malaria durch Chinaalkaloide und ihre theoretischen Grundlagen. *Deutsch. Med. Woch.*, XLIV, 961, 988.

Moritz. F., & Hölzl, H. (1922). Ueber Häufigkeit u. Bedeutung des Vorkommens von *Megastoma entericum* im Darmcanal des Menschen. *Munch. Med. Woch.*, XXXIX, 831.

Moroff, T. (1903). Beitrag zur Kenntniss einiger Flagellaten. *Arch. Protist.*, III, 69.

—— (1906). Untersuchungen über Coccidien. I. *Adelea zonula* nov. sp. *Ibid.*, VIII, 17.

—— (1906a). Sur l'évolution des prétendues coccidies des céphalopodes. *C. R. Acad. Sci.*, CXLII, 652.

—— (1908). Die bei den Cephalopoden vorkommenden *Aggregata*arten als Grundlage einer kritischen Studie über die Physiologie des Zellkernes. *Arch. Protist.*, XI. 1.

—— (1911). Untersuchungen über Coccidien. II. *Klossia vitrina* Moroff. *Ibid.*, XXIII, 51.

Motas, C. (1903). La piroplasmose ovine " carceag." *C. R. Soc. Biol.*, LIV, 1522.

—— (1903a). Sur le rôle des tiques dans le développement de la piroplasmose ovine (*Carceag*). *Ibid.*, LV, 501.

Mott. F. W. (1899). The changes in the central nervous system of two cases of negro lethargy: sequel to Dr. Manson's clinical report. *Brit. Med. Journ.*, II, 1666.

—— (1906). Histological observations on sleeping sickness and other trypanosome infections. *Rep. Sleep. Sickn. Comm. Roy. Soc.*, VII, 3.

Moussu, G., & Marotel, G. (1902). La coccidiose du mouton et son parasite. *Arch. Parasit.*, VI, 82.

Mrázek, A. (1897). Ueber eine neue Sporozoenform aus *Limnodrilus*, *Myxocystis ciliata*. *Sitzungsb. Böhm. Ges. Wiss.*, Math.-Naturw., 1.

—— (1910). Sporozoenstudien. Zur Auffassung der Myxocystiden. *Arch. Protist.*, VIII, 245.

Mühlens, P. (1921). Beobachtungen über das Verhalten der Malariaparasiten in der Anophelesmücke. *Arch. Schiffs.- u.Trop.- Hyg.*, XXV, 58.

Mühlens, P., & Kirschbaum. W. (1921). Parasitologische und klinische Beobachtungen bei künstlichen Malaria- und Rekurrensübertragung. *Zeitschr. Hyg. u. Infekt.*, XCIV, 1.

—— (1924). Weitere parasitologische Beobachtungen bei künstlichen Malariainfektionen von Paralytikern. *Arch. Schiffs.- u. Tropen.-Hyg.*, XXVIII, 131.

Mühlens, P., Weygandt, W., & Kirschbaum. W. (1920). Die Behandlung der Paralyse mit Malaria- und Rekurrensfieber. *Munch. Med. Woch.*, LXVII, 831.

Müller, E. (1889). Elt fynd af *Cercomonas intestinalis* i jejunum från människa. *Nordisk. Med. Ark.*, XXI, 1. Review in *Centralbl. Bakt.*, I. Abt., VIII, 592.

Müller, J. (1841). Ueber eine eigenthümliche krankhafte parasitische Bildung mit specifisch organisirten Samenkörperchen. *Arch. Anat. & Phys.*, 477.

—— (1841a). Krankhafte Hautausschlag mit specifisch organisirten Samenkörperchen (Psorospermien). *Ber. Preuss. Akad. Wiss.*, 212, 246.

Müller, O. F. (1773). *Vermium terrestrium et fluviatilium seu animalium et infusoriorum. helminthicorum et testaceorum, non marinorum, succincta historia*. Havniæ et Lipsiæ.

Munk, F., & Rocha-Lima, H. da (1917). Klinik und Aetiologie des sogen. " Wolhynischen Fiebers " (Werner-Hissche Krankheit). II. Ergebnis des ätiologischen Untersuchungen und deren Beziehungen zur Fleckfieberforschung. *Münch. Med. Woch.*, LXIV, 1422.

MUSGRAVE, W. E., & CLEGG, M. T. (1903). *Trypanosoma* and trypanosomiasis, with special reference to surra in the Philippine Islands. *Bureau Gov. Lab.*, Manila, Bull. No. 5.

————— (1904). Amebas: their cultivation and ætiologic significance. *Dept. Interior, Bureau Gov. Lab., Biol. Lab. Bull.*, No. 18, Part I., 1, Manila.

————— (1906). The cultivation and pathogenesis of amœbæ. *Philippine Journ. Sci.*, I, 909.

MUSLOW, K. (1911). Über Fortpflanzungserscheinungen bei *Monocystis rostrata* n. sp. *Arch. Protist.*, XXII, 20.

[NABARRO, D.] (1907), LAVERAN, A., & MESNIL, F. *Trypanosomes and trypanosomiases.* Translated and much enlarged by Nabarro. London.

NÄGLER, K. (1909). Entwicklungsgeschichtliche Studien über Amöben. *Arch. Protist.*, XV, 1.

NAPIER, L. E. (1921). A new serum test for kala-azar. *Indian Med. Gaz.*, LVI, 238.

————— (1922). A new serum test for kala-azar. *Indian Journ. Med. Res.*, IX, 830.

NASSE, H. (1843). Ueber die eiförmigen Zellen der tuberkelähnlichen Ablagerungen in den Gallengängen der Kaninchen. *Arch. Anat. & Phys.*, 209.

NATTAN-LARRIER, L. (1912). Non-transmission des trypanosomiases de la mère au fœtus. *Bull. Soc. Path. Exot.*, V, 550.

————— (1913). Sur le passage des trypanosomes dans le lait. *Rev. Path. Comp.*, XIII, 282.

————— (1921). Hérédité des infections expérimentales à *Schizotrypanum cruzi.* *Bull. Soc. Path. Exot.*, XIV, 232.

————— (1922). Sur un hémogrégarine de l'homme. (Note préliminaire.) (With comments by Roubaud.) *Ibid.*, XV, 943.

————— (1923). Sur un hémogrégarine de l'homme. (Deuxième note.) *Ibid.*, XVI, 403.

NAWROTZKY, N. N. (1912). Zur Piroplasmoseinfektion der Hunde durch die Schleimhaut des Magendarmtraktes. *Centralbl. Bakt.*, I. Abt., LXVI, 417.

NEAVE, S. (1906). Report of travelling pathologist and naturalist. *2nd Rept., Wellcome Trop. Res. Labs., Khartoum*, 183.

NEGRI, A. (1908). Beobachtungen über Sarcosporidien—I & II. *Centralbl. Bakt.*, I Abt., XLVII, 56, 612.

————— (1910). Beobachtungen über Sarcosporidien—III. *Ibid.*, I. Abt., LV, 373.

————— (1913). Beobachtungen über *Hæmoproteus.* *Ibid.*, I. Abt., LXVIII, 599.

NEIVA, A. (1913). Da transmissão do *Trypanosoma cruzi* pela *Triatoma sordida* Stal. *Brazil-Medico*, XXVII, 309.

————— (1913a). Transmissão do *Trypanosoma cruzi* pelo *Rhipicephalus sanguineus* (Latr.). (Nota prévia.) *Ibid.*, XXVII, 498.

————— (1914). Presença em una localidade do Estado do Rio de um novo transmissor da "Molestia de Chagas" encontrado infectado em condiçoes naturaes. (Nota prévia.) *Ibid.*, XXVIII, 333.

NEIVA, A., DA CUNHA, A., & TRAVASSOS. L. (1914). Contribuiçoes parazitolojicas. *Mem. Inst. O. Cruz*, VI, 180.

[NELIGAN, A. R.] (1913). On the discovery by Dr. A. R. Neligan of leishmania in cutaneous lesions of dogs in Teheran, Persia. *Journ. Trop. Med. & Hyg.*, XVI, 156.

NERESHEIMER, E. (1906). Die Zeugungskreis von *Opalina.* *Sitzungsb. Ges. Morph. Munchen*, XXII, 24.

————— (1907). Die Fortpflanzung der Opalinen. *Arch. Protist.*, IX, Suppt. I. (Festschr. f. R. Hertwig), 1.

————— (1908). Zur Fortpflanzung eines parasitischen Infusors (*Ichthyophthirius*). *Sitzungsb. Ges. Morph. München*, XXIII, 102.

————— (1912). Die Gattung *Trypanoplasma* (Laveran u. Mesnil). Prowazek's *Handb. d. Path. Prot.*, Leipzig, I, 101.

NERESHEIMER, E., & CLODI, C. (1914). *Ichthyophonus hoferi* Plehn u. Mulsow, der Erreger der Taumelkrankheit der Salmoniden. *Arch. Protist.*, XXXIV, 217.

NEUMANN, E. (1866). Kleinere Mittheilungen. 3. Psorospermien im Darmepithel. *Arch. Mikr. Anat.*, II, 512.

NEUMANN, R. O. (1908). Ueber protozoische Parasiten im Blut von Meeresfischen. *Münch. Med. Woch.*, LV, 1460.

————— (1908a). Die Übertragung von *Plasmodium præcox* auf Kanarienvögel durch *Stegomyia fasciata* und die Entwickelung der Parasiten im Magen und der Speicheldrüsen dieser Stechmücke. *Arch. Protist.*, XIII, 23.

————— (1909). Studien über protozoische Parasiten im Blut von Meeresfischen. *Zeitschr. Hyg. u. Infekt.*, LXIV, 1.

————— (1909a). Ueber die Blutparasiten von *Vesperugo* und deren Weiterentwicklung in den Milben der Fledermäuse. *Arch. Protist.*, XVIII, 1.

————— (1909b). *Leishmania tropica* in peripheren Blute bei der Delhibeule. *Centralbl. Bakt.*, I. Abt., LII, 469.

NEVEU-LEMAIRE (1912). *Parasitologie des animaux domestiques.* Paris.

NEWHAM, H. B. (1919). Trypanosomiasis in the East African campaign. *Journ. R. Army Med. Corps*, XXXIII, 299.

NEWHAM, H. B., & ROBERTSON, A. (1922). A case of *Isospora hominis* (Rivolta), Dobell, probably contracted in Durban, South Africa. *Journ. Trop. Med. & Hyg.*, XXV, 344.

NICLOT, BAUR, J., MONIER-VINARD, & BÉGUET, M. (1918). *Le paludisme*. Macedoine, 1915-1916-1917.

NICOLLE, C. (1907). Sur une piroplasmose nouvelle d'un rongeur. *C. R. Soc. Biol.*, LXIII, 213.

—— (1908). Sur trois cas d'infection splénique infantile à corps de Leishman observés en Tunisie. *Arch. Inst. Pasteur Tunis*, III, 3.

—— (1908a). Nouvelles acquisitions sur le kala-azar· cultures; inoculation au chien; éthologie. *C. R. Acad. Sci.*, CXLVI, 498.

—— (1908b). Culture du parasite du bouton d'orient. *Ibid.*, CXLVI, 842.

—— (1908c). Culture des corps de Leishman isolés de la rate dans trois cas d'anémie splénique infantile. *Bull. Soc. Path. Exot.*, I, 121.

—— (1908d). Reproduction expérimentale du kala-azar chez le chien. Origine canine probable de cette infection. *Ibid.*, I, 188.

—— (1909). Le kala-azar infantile. *Ann. Inst. Pasteur*, XXIII, 361.

—— (1909a). Quelques données nouvelles relatives au kala-azar infantile. *Bull. Soc. Path. Exot.*, II, 457.

—— (1910). Quelques données nouvelles relatives au kala-azar infantile. *Ibid.*, III, 431.

—— (1914). Chronique du kala-azar en Tunisie. *Ibid.*, VII, 479.

—— (1921). Chronique du kala-azar en Tunisie. *Arch. Inst. Pasteur Afrique du Nord*, I, 33.

—— (1925). Chronique du kala-azar en Tunisie. *Arch. Inst. Pasteur Tunis*, XIV, 136.

NICOLLE, C., & ANDERSON, C. (1923). Recherches expérimentales sur le mode de transmission du kala-azar. *Ibid.*, XII, 168.

—— (1924). Recherches expérimentales sur le mode de transmission du kala-azar. Deuxième mémoire. *Ibid.*, XIII, 155.

—— (1925). Recherches expérimentales sur le mode de transmission du kala-azar. *Ibid.*, XIV, 264.

NICOLLE, C., & BLAIZOT, L. (1912). Virulence des cultures de *Leishmania infantum*. Sensibilité du chacal au virus du kala-azar tunisien. *Bull. Soc. Path. Exot.*, V, 721.

NICOLLE, C., BLANC, G., & LANGERON, M. (1920). Recherches expérimentales sur le rôle du gecko (*Tarentola mauritanica*) dans l'étiologie du bouton d'orient. (Note préliminaire.) *Ibid.*, XIII, 508.

NICOLLE, C., & COMTE, C. (1908). Origine canine du kala-azar. *C. R. Acad. Sci.*, CXLVI, 789.

—— —— (1908a). Origine canine du kala-azar. *Arch. Inst. Pasteur Tunis*, III, 59, 109.

—— —— (1908b). Sur un trypanosome d'une chauve-souris. *Ibid.*, III, 69.

—— —— (1909). Contribution à l'étude de *Trypanosoma vespertilionis*. *Ibid.*, IV, 202.

—— —— (1910). Kala-azar expérimental du chien. Quelques observations nouvelles et quelques faits nouveaux. Immunité par première atteinte après guérison complète. *Ibid.*, V, 103.

NICOLLE, C., COMTE, C., & CONSEIL, E. (1909). Transmission expérimentale du typhus exanthématique par le pou du corps. *C. R. Acad. Sci.*, CXLIX, 486.

NICOLLE, C., COMTE, C., & MANCEAUX, L. (1908). Recherches sur le kala-azar entreprises à l'Institut Pasteur de Tunis. (Nouvelle série d'expériences.) *Arch. Inst. Pasteur Tunis*, III, 97.

NICOLLE, C., & MANCEAUX, L. (1908). Sur une infection à corps de Leishman (ou organismes voisins) du gondi. *C. R. Acad. Sci.*, CXLVII, 763.

—— —— (1909). Sur un protozoaire nouveau du gondi. *Ibid.*, CXLVIII, 369.

—— —— (1910). Reproduction expérimentale du bouton d'orient chez le chien. Origine canine possible de cette infection. *Ibid.*, CL, 889.

—— —— (1910a). Recherches sur le bouton d'orient. Cultures, reproduction expérimentale, immunisation. *Ann. Inst. Pasteur*, XXIV, 673.

NICOLLE, C., & SICRE, A. (1908). Recherches sur le bouton d'orient. 2. Isolement et culture du parasite du bouton d'orient. *Arch. Inst. Pasteur Tunis*, III, 117.

—— (1908a). Reproduction expérimentale du bouton d'orient chez le singe (*Macacus sinicus*). *C. R. Soc. Biol.*, LXIV, 1096.

NICOLLE, M.. & ADIL-BEY (1899). Première note sur la malaria des bovidés. *Ann. Inst. Pasteur*, XIII, 337.

NICOLLE, M., & MESNIL, F. (1906). Traitement des trypanosomiases par les couleurs de benzidine. 1re partie. Etude chimique. *Ibid.*, XX, 417.

NIESCHULZ, O. (1921). Bijdrage tot de kennis van eenige vogeltrypanosomen. (Voorl. mededeel.) *Tijdschr. Diergeneesk.*, XLVIII, 569.

—— (1921a). Beiträge zur Kenntnis der Gattung *Eimeria*. I. Über das Taubencoccid. *Arch. Protist.*, XLIV, 71.

—— (1921b). Over de betrekking tusschen het duiven- en kippen-coccidium. *Tijdschr. Diergeneesk.*, XLVIII, 707.

—— (1922). Ueber die Benennung des Schweincoccids. *Centralbl. Bakt.*, I. Abt., LXXXVIII, 379.

NIESCHULZ, O. (1922a). Zur Kenntnis einiger Vogeltrypanosomen. *Arch. Protist.*, XLV, 241.
—— (1922b). Über Entamöben des Hausrindes. *Ibid.*, XLV, 410.
—— (1923). Een geval van Giardia-infectie bij een kalf. *Tijdschr. Diergeneesk.*, L, 733.
—— (1923a). Over de Darmamœben van het varken. *Ibid.*, L, 736.
—— (1923b). *Giardia capræ* n. sp. en *Entamœba* sp., nieuwe darmparasieten van de geit. *Ibid.* L, 780.
—— (1923c). Ueber Hasenkokzidien (*Eimeria leporis* n. sp.). *Deutsch. Tierärztl. Woch.*, XXXI. 245.
—— (1924). Ein weiterer Fall von *Eimeria canis* Wenyon. *Berl. Tierärztl. Woch.*, XL, 220.
—— (1924a). Over een geval van Eimeria-infectie bij een kat (*Eimeria felina* n. sp.). *Tijdschr. Diergeneesk.*, LI, 129.
—— (1924b). Über *Entamœba debliecki* mihi, eine Darmamöbe des Schweines. *Arch. Protist.*, XLVIII, 365.
—— (1924c). Amöben aus dem Zahnbelag von Pferden. *Arch. Tierheilk.*, LI, 41.
—— (1924d). Zur Morphologie der Kulturformen einer *Herpetomonas* aus *Euphorbia cereiformis*. *Centralbl. Bakt.*, II. Abt., LXI, 311.
—— (1924e). Über den Bau von *Giardia capræ* mihi. *Arch. Protist.*, XLIX, 278.
—— (1925). Zur Verbreitung von *Isospora*-Infektionen bei Hunden und Katzen in den Niederlanden. *Centralbl. Bakt.*, I. Abt., XCIV, 137.:
—— (1925a). Über die Entwicklung des Taubencoccids *Eimeria pfeifferi* (Labbé, 1896). *Arch. Protist.*, LI, 479.
NOC, F. (1908). Un cas de dysenterie à *Balantidium* chez le *Macacus cynomolgus*. *C. R. Soc. Biol.*, LXIV, 878.
—— (1909). Recherches sur la dysenterie amibienne en Cochinchine. *Ann. Inst. Pasteur*, XXIII, 177.
—— (1909a). Observations sur le cycle évolutif de *Lamblia intestinalis*. *Bull. Soc. Path. Exot.*, II, 93.
—— (1920). Nouveau cas de coccidiose intestinale humaine à *Isospora*. *Ibid.*, XIII, 785.
—— (1922). Formes hémogrégariniennes observées dans un cas d'anémie pernicieuse autochtone. [With comments by Roubaud.] *Ibid.*, XV, 936.
NOC, F., & STÉVENEL, L. (1911). Présence à la Martinique de *Leptomonas davidi* Lafont. *Ibid.*, IV, 461.
NOCARD & ALMY (1901). Une observation de piroplasmose canine. *Bull. Soc. Centr. Méd. Vét.*, LV, 192.
NOCARD & MOTAS (1902). Contribution à l'étude de la piroplasmose canine. *Ann. Inst. Pasteur*, XVI, 257.
NÖLLER, W. (1912). Über Blutprotozoen einheimischer Nagetiere und ihre Übertragung. *Berlin. Klin. Woch.*, XLIX, i, 524.
—— (1912a). *Entamœba aulastomi* nov. spec., eine neue parasitische Amöbe aus dem Pferdeegel (*Aulastomum gulo* Moq.-Tand.). *Arch. Protist.*, XXIV, 195.
—— (1912b). Über eine neue Schizogonie von *Lankesterella minima* Chaussat (=*L. ranarum* Lank.). *Ibid.*, XXIV, 201.
—— (1912c). Die Blutprotozoen des Hamsters (*Cricetus frumentarius* Pall.) und ihre Übertragung. (Vorl. Mitt.) *Ibid.*, XXV, 377.
—— (1912d). Die Übertragungsweise der Rattentrypanosomen durch Flöhe. *Ibid.*, XXV, 386.
—— (1913). Die blutsaugenden Insekten als Krankheitsüberträger. *Monatsh. Prakt. Tierheilk.*, XXV, 68.
—— (1913a). Die Blutprotisten des Wasserfrosches und ihre Übertragung. (Vorl. Mitt.) *Arch. Protist.*, XXVIII, 313.
—— (1913b). Die Blutprotozoen des Wasserfrosches und ihre Übertragung. I. Teil. *Ibid.*, XXXI, 169.
—— (1914). Die Übertragungsweise der Rattentrypanosomen. II. Teil. *Ibid.*, XXXIV, 295.
—— (1916). Die Übertragung des *Trypanosoma theileri* Laveran, 1902. (Vorl. Mitt.) *Berlin. Tierärztl. Woch.*, XXXII, 457.
—— (1917). Blut- und Insektenflagellatenzüchtung auf Platten. *Arch. Schiffs.- u. Trop.-Hyg.*, XXI, 53.
—— (1919). Beitrag zur Kenntnis des Schaftrypanosomas. (Vorl. Mitt.) *Ibid.*, XXIII, 99.
—— (1919a). Zur Verbreitung des Schaftrypanosomes bei heimischen Schafen. *Deutsch. Tierärztl. Woch.*, XXVII, 327.
—— (1920). Die Toxoplasmen. Prowazek's *Handb. d. Path. Prot.*, II, 907.
—— (1920a). Die neueren Ergebnisse der *Hæmoproteus*-Forschung. *Arch. Protist.*, XLI, 149.
—— (1920b). Kleine Beobachtungen an parasitischen Protozoen. (Zugleich vorläufige Mitteilung über die Befruchtung und Sporogonie von *Lankesterella minima* Chaussat.) *Ibid.*, XLI, 169.
—— (1920c). Neuere Forschungen auf dem Gebiete der Trypanosomenzüchtung. *Arch. Schiffs.-u. Trop.-Hyg.*, XXIV, 168.

NÖLLER, W. (1920d). Neuere Forschungen auf dem Gebiete der Blutparasiten unter den Sporozoen. *Ibid.*, XXIV, 353.

—— (1921). Ueber einige wenig bekannte Darmprotozoen des Menschen und ihre nächsten Verwandten. *Ibid.*, XXV, 35.

—— (1921a). Die Bekämpfung der parasitären Erkrankungen unserer Haustiere. *Deutsch. Landwirtsch. Ges., Jahrb., 1921*, 1.

—— (1922). Die wichtigsten parasitischen Protozoen des Menschen und der Tiere. I. Teil. Ostertag, Wolffhügel & Nöller's *Tier. Parasit. d. Haus- u. Nutzt.*, 1.

—— (1923). Zur Kenntnis eines Nierencoccids. Der Entwicklungskreis des Coccids der Wasserfroschniere. *Isospora lieberkühni* (Labbé 1894). *Arch. Protist.*, XLVII, 101.

—— (1925). Der Nachweis des Überträgers des gemeinen Rindertrypanosomas, *Trypanosoma theileri*, mit Hilfe des Kulturverfahrens. *Centralbl. Bakt.*, Ref. LXXIX, 133.

NÖLLER, W., & BUTTGEREIT, F. (1923). Über ein neues parasitisches Protozoon der Haustaube (*Octomitus columbæ* nov. spec.). *Ibid.*, Ref. LXXV, 239.

NÖLLER, W., & FRENZ, O. (1922). Zur Kenntnis des Ferkelkokzids und seiner Wirkung. *Deutsch. Tierärztl. Woch.*, XXX, 1.

NÖLLER, W., KROSZ, K., & ARNDT, A. (1921). Die Rhizopodenfauna des Pferdekotes und des Strassenstaubes in ihren Beziehungen zu Darmpassanten des Menschen. *Arch. Schiffs.- u. Trop.-Hyg.*, XXV. 114.

NÖLLER, W., & KUCHLING, M. (1923). Zur Züchtung des Schaftrypanosomas und der Schaflausrickettsia aus dem Schafblute. *Berlin. Tierärztl. Woch.*, XXXIX, 197.

NÖLLER, W., SCHÜRJOHANN, S., & VORBRODT, K. (1922). Zur Kenntnis der Ziegen- und Schafkokzidiose. (Vorläuf. Mitt.) *Ibid.*, XXXVIII, 193.

NOGUCHI, H. (1924). Action of certain biological, chemical, and physical agents upon cultures of Leishmania; some observations on plant and insect herpetomonads. *Proc. Internat. Conf. on Health Problems in Trop. Amer.*, Boston, Mass., 455.

—— (1924a). [Discussion following paper by Mühlens.] *Ibid.*, 553.

NOGUCHI, H.. & LINDENBERG, A. (1925). The isolation and maintenance of leishmania on the medium employed for the cultivation of organisms of the leptospira group of spirochetes. *Amer. Journ. Trop. Med.*, V, 63.

NOVY, F. G. (1906). The trypanosomes of tsetse flies. *Journ. Infect. Dis.*, III, 394.

—— (1908). Successful canine infection with cultures of *Leishmania infantum* (C. Nicolle). *Journ. Amer. Med. Assoc.*, LI, 1423.

NOVY, F. G., & MACNEAL. W. J. (1904). Trypanosomes and bird malaria. *American Medicine*, Philadelphia, VIII, 932.

—— —— (1904a). On the filtration of trypanosomes. *6th Rept. Michigan Acad. Sci.*, 180.

—— —— (1905). On the trypanosomes of birds. *Journ. Infect. Dis.*, II, 256.

NOWLIN, N. (1917). *Endamœba buccalis*. I. Its multiplication and periodicity. *Journ. Parasit.*, III. 143.

NUTTALL, G. H. F. (1901). On the question of priority with regard to certain discoveries upon the ætiology of malarial diseases. *Quart. Journ. Micro. Sci.*, XLIV, 429.

—— (1904). Canine piroplasmosis. *Journ. Hyg.*, IV. 219.

—— (1908). Piroplasmosis. The Harben Lectures. III. *Journ. R. Inst. Pub. Health.*, XVI, 513.

—— (1909). The transmission of *Trypanosoma lewisi* by fleas and lice. *Parasitology*, I, 296.

—— (1910). On Hæmatozoa occurring in wild animals in Africa. I. *Piroplasma rossi* n. sp. and *Hæmogregarina canis adusti* n. sp. found in the jackal. *Ibid.*, III, 108.

—— (1912). Note on *Rossiella rossi* (Nuttall, 1910) occurring in the jackal of British East Africa. *Ibid.*, V, 61.

—— (1913). The Herter Lectures. III. Piroplasmosis. *Ibid.*, VI, 302.

NUTTALL, G. H. F., FANTHAM. H. B., & PORTER, A. (1909). Observations on *Theileria parva*, the parasite of East Coast fever of cattle. *Ibid.*, II, 325.

NUTTALL, G. H. F., & GRAHAM-SMITH, G. S. (1905). Canine piroplasmosis—II. *Journ. Hyg.*, V, 237.

—— —— (1906). Canine piroplasmosis. V. Further studies on the morphology and life-history of the parasite. *Ibid.*, VI, 586.

—— —— (1907). Canine piroplasmosis. VI. Studies on the morphology and life-history of the parasite. *Ibid.*. VII, 232.

—— —— (1908). The mode of multiplication of *Piroplasma bovis* and *P. pitheci* in the circulating blood compared with that of *P. canis*, with notes on other species of *Piroplasma*. *Parasitology*, I, 134.

—— —— (1908a). The development of *Piroplasma canis* in culture. *Ibid.*, I, 243.

—— —— (1909). *Theileria parva*: attempts at cultivation. *Ibid.*, II, 208.

—— —— (1909a). Note on attempts to infect the fox and the jackal with *Piroplasma canis*. *Ibid.*, II, 211.

NUTTALL, G. H. F., & HADWEN, S. (1909). The successful drug treatment of canine piroplasmosis, together with observations upon the effect of the drug on *Piroplasma canis*. *Ibid.*, II, 156.

NUTTALL, G. H. F. & HADWEN, S. (1909a). Further experiments upon the drug treatment of canine piroplasmosis. *Ibid.*, II, 229.

NUTTALL, G. H. F., & HINDLE, E. (1913). Conditions influencing the transmission of East Coast fever. *Ibid.*, VI, 321.

NUTTALL, G. H. F., & STRICKLAND, C. (1910). Die Parasiten der Pferdepiroplasmose resp. der "Biliary fever." *Centralbl. Bakt.*, I. Abt., LVI, 524.

—— —— (1912). On the occurrence of two species of parasites in equine "piroplasmosis" or "biliary fever." *Parasitology*, V, 65.

OCHMANN, R. (1905). Trypanosomiasis beim Schweine. *Berlin. Tierärztl. Woch.*, 337.

O'CONNOR, F. W. (1919). Intestinal Protozoa found during acute intestinal conditions amongst members of the Egyptian Expeditionary Force, 1916-17. *Parasitology*, XI, 239.

—— (1920). A preliminary note on two intestinal parasites of pigs. *Med. Journ. Australia*, II, 337.

OEHLER, R. (1913). Ueber die Gewinnung reiner Trypanosomenstämme durch Einzellenüber-tragung. *Centralbl. Bakt.*, I. Abt., LXVII, 569.

O'FARRELL, W. R. (1913). Hereditary infection, with special reference to its occurrence in Hyalomma ægyptium infected with Crithidia hyalommæ. *Ann. Trop. Med. & Parasit.*, VII, 545.

OGAWA, M. (1913). Sur un trypanosome de Triton pyrrhogaster. *C. R. Soc. Biol.*, LXXV, 268.

OHIRA, T., & NOGUCHI, H. (1917). The cultivation of Trichomonas of the human mouth (Tetra-trichomonas hominis). *Journ. Exp. Med.*, XXV, 341.

OHLMACHER, A. P. (1893). Myxosporidia in the common toad, with preliminary observations on two chromophile substances in their spores. *Journ. Amer. Med. Assoc.*, XX, 561.

OLIVER, J. (1922). Spontaneous chronic meningo-encephalitis of rabbits. *Journ. Infect. Dis.*, XXX, 91.

OLLWIG, H., & MANTEUFEL, P. (1912). Die Babesien. Prowazek's *Handb. d. Path. Prot.*, II, 517.

PACHINGER, A. (1886). Mittheilung über Sporozoen. *Zool. Anz.*, IX, 471.

PAES, S. (1924). Sur une plasmodie du sang des chèvres. *Rev. Méd. Angola*, Num. Espec. I° Cong. Med. Trop. Africa Oc., IV, 441.

PARISI, B. (1910). Su alcuni flagellati endoparassiti. *Arch. Protist.*, XIX, 232.

PARISOT, J., & SIMONIN, P. (1921). Gangrène pulmonaire et Trichomonas. *C. R. Soc. Biol.*, LXXXV, 1077.

PARKER, R. R. (1923). Transmission of Rocky Mountain spotted fever by the rabbit tick Hæma-physalis leporispalustris Packard. *Amer. Journ. Trop. Med.*, III, 39.

PARROT, L. (1919). Trois observations de bouton d'orient avec des réflexions sur les circonstances de la contamination. *Bull. Soc. Path. Exot.*, XII, 607.

PATTEN, M. W. (1921). The life-history of an amicronucleate race of Didinium nasutum. *Proc. Soc. Exp. Biol. & Med.*, XVIII, 188.

PATTON, W. H. (1895). The name of the southern or splenic cattle-fever parasite. *Amer. Natural.*, XXIX, 498.

PATTON, W. S. (1906). On a parasite found in the white corpuscles of the blood of palm squirrels. *Sci. Mem. Off. Med. & San. Dep. Gov. India*, New Series, No. 24.

—— (1907). Preliminary report on the development of the Leishman-Donovan body in the bed bug. *Ibid.*, No. 27.

—— (1907a). The development of the Leishman-Donovan parasite in Cimex rotundatus. *Ibid.*, No. 31.

—— (1907b). The development of the Leishman-Donovan parasite in Cimex rotundatus. Append. *Ann. Rept. Bact. Sect. King Inst. Prev. Med. Guindy, 1907.* Madras.

—— (1908). The hæmogregarines of mammals and reptiles. *Parasitology*, I, 318.

—— (1908a). The life-cycle of a species of Crithidia parasitic in the intestinal tract of Gerris fossarum Fabr. *Arch. Protist.*, XII, 131.

—— —— (1908b). Herpetomonas lygœi. *Ibid.*, XIII, 1.

—— (1908c). Annual report upon the work of the bacteriological section of the King Institute of Preventive Medicine, Guindy, for the year 1907. Madras.

—— (1909). The life-cycle of a species of Crithidia parasitic in the intestinal tracts of Tabanus hilarius and Tabanus sp. ?. *Arch. Protist.*, XV, 333.

—— (1910). Preliminary report on a new piroplasm (Piroplasma gibsoni sp. nov.) found in the blood of the hounds of the Madras hunt and subsequently discovered in the blood of the jackal, Canis aureus. *Bull. Soc. Path. Exot.*, III, 274.

—— (1910a). Rhynchomonas luciliæ nov. gen., nov. spec. A new flagellate parasitic in the Malpighian tubes of Lucilia serenissima Walk. *Ibid.*, III, 300, 433.

—— (1910b). Experimental infection of the Madras bazaar fly, Musca nebulo Fabr., with Herpeto-monas muscæ domesticæ Burnett. *Ibid.*, III, 264.

—— —— (1912). Preliminary report on an investigation into the aetiology of oriental sore in Cambay. *Sci. Mem. Off. Med. & San. Dep. Gov. India.* New Series, No. 50.

PATTON, W. S. (1912a). The development of the parasite of Indian kala-azar (*Herpetomonas donovani*, Laveran and Mesnil) in *Cimex rotundatus* Sign. and in *Cimex lectularius* Linn., with some observations on the behaviour of the parasite in *Conorrhinus rubrofasciatus*, de Geer. *Ibid.*, No. 53.

—— (1912b). Studies on the flagellates of the genera *Herpetomonas*, *Crithidia*, and *Rhynchoidomonas*. No. 1.—The morphology and life-history of *Herpetomonas culicis*, Novy, MacNeal, & Torrey. *Ibid.*, No. 57.

—— (1913). Is kala-azar in Madras of animal origin ? *Indian Journ. Med. Res.*, I, 185.

—— (1914). The examination of the peripheral blood of 84 patients suffering from kala-azar at the General Hospital, Madras, during the period from 15th June, 1912, to 15th July, 1913. *Ibid.*, II, 492.

—— (1919). Note on the ætiology of oriental sore in Mesopotamia. *Bull. Soc. Path. Exot.*, XII, 500.

—— (1920). Some notes on the arthropods of medical and veterinary importance in Mesopotamia, and on their relation to disease. Part IV. *Indian Journ. Med. Res.*, VIII, 245.

—— (1921). Studies on the flagellates of the genera *Herpetomonas*, *Crithidia*, and *Rhynchoidomonas*. No. 7.—Some miscellaneous notes on insect flagellates. *Ibid.*, IX, 230.

—— (1922). Some reflections on the kala-azar and oriental sore problems. *Ibid.*, IX, 496.

PATTON, W. S., LA FRENAIS, H. M., & RAO, S. (1921). Studies on the flagellates of the genera *Herpetomonas*, *Crithidia*, and *Rhynchoidomonas*. No. 8.—Note on the behaviour of *Herpetomonas tropica*, Wright, the parasite of cutaneous Herpetomonas (oriental sore) in the bed bug, *Cimex hemiptera*, Fabr. No. 9.—Note on the behaviour of *Herpetomonas donovani*, Laveran and Mesnil, in the bed bug, *Cimex hemiptera*, Fabricius. *Ibid.*, IX, 240, 252.

PATTON, W. S., & RAO, S. (1921). Studies on the flagellates of the genera *Herpetomonas*, *Crithidia*, and *Rhynchoidomonas*. No. 5.—The morphology and life-history of *Herpetomonas pulicis* sp. nov., parasitic in the alimentary tract and Malpighian tubes of *Pulex irritans* L. *Ibid.*, VIII, 621.

PATTON, W. S., & STRICKLAND, C. (1908). A critical review of the relation of blood-sucking invertebrates to the life-cycles of the trypanosomes of vertebrates, with a note on the occurrence of a species of *Crithidia*, *C. ctenophthalmi*, in the alimentary tract of *Ctenophthalmus agyrtes*, Heller. *Parasitology*, I, 322.

PAULICKI, A. (1872). Beiträge zur vergleichenden pathologischen Anatomie aus dem Hamburger Zoologischen Garten. *Mag. ges. Theirheilk.*, XXXVIII, 1.

PAVONI, G. (1915). Contributo allo studio della infezione sperimentale del *Mus musculus* con *Leishmania tropica* e *infantum*. *Pathologica*, VII, 114.

PEARCE, L. (1916). *Klossiella* infection of the guinea-pig. *Journ. Exp. Med.*, XXIII, 431; *Studies, Rockefeller Inst. Med. Res.*, XXV, 189.

—— (1921). Studies on the treatment of human trypanosomiasis, with tryparsamide (the sodium salt of N-phenylglycineamide-*p*-arsonic acid). *Journ. Exp. Med.*, XXXIV, Suppl. 1.

PÉCAUD, G. (1909). Contribution à l'étude de l'étiologie de la souma. *Bull. Soc. Path. Exot.*, II, 530.

PEDROSO, A. (1910). Cultura de *Leishmania tropica*. *Arch. Soc. Med. e Cirurg. S. Paulo*, I.

PEDROSO, A. M. (1913). Leishmaniose local do Cão. *An. Paulist. Med. e Cir.*, I, 33.

PENARD, E. (1914). Sur quelques tentaculifères muscicoles. *Arch. Protist.*, XXXIV, 277.

—— (1922). *Etudes sur les infusoires d'eau douce*. Geneva.

PENFOLD, W. J., WOODCOCK, H. M., & DREW, A. H. (1916). The excystation of *Entamœba histolytica* (*tetragena*) as an indication of the vitality of the cysts. *Brit. Med. Journ.*, I, 714.

PENNING, C. A. (1899). Over het voorkomen van anæmia perniciosa infectiosa of wel surra onder de paarden in Nederlandsch-Indië. *Veearts. Blad. Ned.-Indie*, Batavia, XII, 123.

—— (1900). Verdere waarnemingen betreffende surra in Nederlandsch- Indië. *Ibid.*, XIII, 25.

PENTIMALLI, F. (1923). Flagellati del genere " *Trichomonas* " nel sangue circolante dell' uomo. *Annali d'Igiene*, XXXIII, 309.

PÉRARD, C. (1924). Recherches sur les coccidies et les coccidioses du lapin. *C. R. Acad. Sci.*, CLXXVIII, 2131.

—— (1924a). Recherches sur les coccidies et les coccidioses du lapin. *Ann. Inst. Pasteur*, XXXVIII, 953.

PEREKROPOFF, G. (1914). Ueber Kulturen der Plasmodien des tropischen Fiebers (*Malaria tropica*). *Arch. Protist.*, XXXV, 139.

PEREZ, C. (1903). Le cycle évolutif de l'*Adelea mesnili*, coccidie cœlomique parasite d'un lepidoptère. *Ibid.*, II, 1.

—— (1908). Sur *Dubosquia legeri*, microsporidie nouvelle parasite du *Termes lucifugus*, et sur la classification des microsporidies. *C. R. Soc. Biol.*, LXV, 631.

PERRONCITO, E. (1876). Nuovo caso di psorospermosi intestinale in una gallina. *Ann. R. Acad. Agric. Torino*, XIX, 77.

—— (1882). *I parassiti dell' uomo e degli animali utili. Delle più comuni malattie da essi prodotte.* Milan.

PERRONCITO, E. (1901). *I parassiti dell' uomo e degli animali utili e le più comuni malattie da essi prodotte*. Milan.

PERRY, H. M. (1922). Some observations on the occurrence of *Leishmania* in the intestinal tissues in Indian kala-azar, on the pathological changes occasioned by their presence, and on their possible significance in this situation. *Journ. R. Army Med. Corps,* XXXIX, 323.

PERTY, M. (1852). *Zur Kenntnis kleinster Lebensformen*. Berne.

PESTANA, B. R. (1910). O nambyuvu. *Rev. Med. S. Paulo*, No. 22, 423.

PETRIE, G. F. (1905). Observations relating to the structure and geographical distribution of certain trypanosomes. *Journ. Hyg.*, V, 191.

PETTIT, A. (1916). Sur un sporozaire parasite du cobaye appartenant au genre *Klossiella* Smith et Johnson. *C. R. Soc. Biol.*, LXXIX, 168.

PETZETAKIS, M. (1923). La broncho-amibiase. Bronchites amibiennes pures sans abcès. Présence de l'*Amœba histolytica* dans les crachats. *Bull. & Mém. Soc. Méd. Hôpit. Paris*, XXXIX, 1229.

—— (1923*a*). L' " amibiase rénale." Un cas de néphrite amibienne. Action curative et diurétique de l'emétine. *Ibid.*, XXXIX, 1233.

—— (1923*b*). Bronchites amibiennes (nouveau cas) avec présence d'amibes vivantes dans les crachats sans abcès des poumons et du foie. *Ibid.*, XXXIX, 1431.

PFEIFFER, E. (1905). Über trypanosomenähnliche Flagellaten im Darm von *Melophagus ovinus*. *Zeitschr. Hyg. u. Infekt.*, I, 324.

PFEIFFER, L. (1890). *Die Protozoen als Krankheitserreger*. Jena.

—— (1890*a*). Vergleichende Untersuchungen über Schwärmsporen und Dauersporen bei den Coccidieninfektionen und bei Intermittens. *Fortschr. Med.*, VIII, 939.

—— (1891). *Die Protozoen als Krankheitserreger*. 2nd edition.

—— (1895). *Die Protozoen als Krankheitserreger*. Nachträge, Jena. 11.

PFEIFFER, R. (1892). *Beiträge zur Protozoenforschung. I. Die Coccidienkrankheit der Kaninchen*. Berlin.

PHEAR, A. G. (1920). Medical experiences in Macedonia and the Caucasus. *Proc. Roy. Soc. Med.*, XIII, Occ. Lect., 57.

PHISALIX, [M.] (1923). Coccidiose intestinale de la vipère aspic à *Cyclospora viperæ* n. sp. *Bull. Soc. Path. Exot.*, XVI, 637.

—— (1924). Note complémentaire sur *Cyclospora viperæ* coccidie parasite de l'intestin de la vipère aspic. *Ibid.*, XVII, 559.

—— (1924*a*). Coccidiose intestinale de *Vipera berus* à *Cyclospora babaulti*, nov. sp. *Ibid.*, XVII, 868.

—— (1924*b*). *Cyclospora tropidonoti*, nov. spec., coccidie intestinale de la couleuvre à collier. *Ibid.*, XVII, 871.

PHOTINUS, G. T. (1920). Un nouveau traitement du bouton d'orient (de Crète) par des injections locales de chlorhydrate d'émétine. *Ibid.*, XIII, 290.

PIANA, G. P. (1876). Ricerche sopra una epizoozia dei gallinacei osservata nella Provincia di Bologna. *Gaz. Med. Vet.*, VI, 257.

PIANA, G. P., & GALLI-VALERIO, B. (1895). Su di un' infezione del cane con parassiti endoglobulari nel sangue. *Moderno Zooiatro*, No. 9, 163.

PIANESE, G. (1901). Ueber ein Protozoon des Meerschweinchens. *Zeitzchr. Hyg. u. Infekt.*, XXXVI, 350.

PINTO, C. (1919). Protozoarios parasitos do homem verificados no Estado do Paraná. *Brazil-Medico*, XXXIII, 329.

—— (1923). O *Triatoma brasiliensis* Neiva transmitte o " *Trypanosoma cruzi* " Chagas. *Ibid.*, XXXVII, i, 73.

—— (1923*a*). Transmissão dos Protozoarios. *Sciencia Medica*, I, 17.

—— (1924). Sobre um reduvideo transmissor do *Trypanosoma cruzi*. *Ibid.*, II, 426.

PINTO, C., & PACHECO, G. (1925). Sur la présence de l'*Isospora belli* Wenyon (1923) au Brésil. *C. R. Acad. Sci.*, XCIII, 1258.

PITTALUGA, G. (1912). Ein neuer Blutparasit der afrikanischer Schildkröte, *Clemmys africana*, " *Hæmoproteus cajali*," n. sp. *Centralbl. Bakt.*, I. Abt., LXIII, 241.

—— (1914). Kala-azar infantile e leishmaniosi in Ispagna. *Pathologica*, VI, 121.

PITTALUGA, A., & BUEN, S. (1917). Nota sobre los dipteros del género *Phlebotomus* en España. *Bol. Inst. Nac. Hig. Alfonso XIII*, XIII, 137.

PIXELL-GOODRICH, L. M. (1914). The sporogony and systematic position of the Aggregatidæ. *Quart. Journ. Micro. Sci.*, LX, 159.

PLANER, J. (1854). Ueber das Vorkommen von Pigment im Blute. *Zeitschr. K. K. Ges. Aerzte Wien*, X, i, 127.

PLATE, L. (1914). Brief note on *Toxoplasma pyrogenes*, Castellani, 1913. *Journ. Trop. Med. & Hyg.*, XVII, 98.

PLEHN, M. (1903). *Trypanoplasma cyprini* nov. sp. *Arch. Protist.*, III, 175.

PLIMMER, H. G. (1912). The President's address—On certain blood parasites. *Journ. R. Micro. Soc.*, 133.

PLIMMER, H. G. (1912a). On the blood parasites found in animals in the Zoological Gardens during the four years 1908-1911. *Proc. Zool. Soc. London*, 406.

—— (1914). Report on the deaths which occurred in the Zoological Gardens during 1913, together with a list of the blood parasites found during the year. *Ibid.*, 181.

—— (1915). Report on the deaths which occurred in the Zoological Gardens during 1914, together with a list of the blood parasites found during the year. *Ibid.*, 123.

—— (1916). Report on the deaths which occurred in the Zoological Gardens during 1915, together with a list of the blood parasites found during the year. *Ibid.*, 77.

—— (1916a). Notes on the genus *Toxoplasma*, with a description of three new species. *Proc. Roy. Soc.*, B, LXXXIX, 291.

PLIMMER, H. G., & BRADFORD, J. R. (1899). A preliminary note on the morphology and distribution of the organism found in the tsetse-fly disease. *Ibid.*, LXV, 274.

POCHE, F. (1903). Ueber zwei neue in Siphonophoren vorkommende Flagellaten nebst Bemerkungen über die Nomenclatur einiger verwandter Formen. *Arb. Zool. Inst. Wien.*, XIV, 307.

PODWYSSOZKI, W. W. (1890). Studien über Coccidien. I. Über das Vorkommen der Coccidien in Hühnereiern im Zusammenhange mit der Frage über die Aetiologie der Psorospermosis. *Centralbl. Allg. Path. u. Path. Anat.*, I, 153.

—— (1894). Entwickelungsgeschichte des *Coccidium oviforme* im Zusammenhange mit der Lehre von den Krebsparasiten. *Centralbl. Bakt.*, I. Abt., XV, 480.

—— (1895). Zur Entwickelungsgeschichte des *Coccidium oviforme* als Zell-Schmarotzers. *Bibl. Medica.* Abt. D, ii, Hft. 4 [Part 1], 1.

POISSON, R. (1924). Sur quelques microsporidies parasites d'arthropodes. *C. R. Acad. Sci.*, CLXXXVIII, 664.

—— (1925). *Leptomonas naucoridis* n. sp. parasite intestinal de *Naucoris maculatus* Fabr. *Ann. Parasit. Hum. & Comp.*, III, 28.

PONS, R. (1925). Coccidiose intestinale humaine à *Isospora belli* (Wenyon, 1922). Considérations générales. *Bull. Soc. Path. Exot.*, XVIII, 570.

PONSELLE, A. (1913). Culture *in vitro* du "*Trypanoplasma varium*" Léger. *C. R. Soc. Biol.*, LXXIV, 685.

—— (1919). *Hexamitus intestinalis*, parasite habituel de l'intestin des batraciens, trouvés dans le sang de *Rana esculenta*. *Ibid.*, LXXXII, 23.

—— (1923). La culture de *Trypanosoma inopinatum* trypanosome pathogène de la grenouille. *Ann. Parasit. Hum. & Comp.*, I, 155.

—— (1923a). Essai d'immunisation contre les trypanosomes pathogènes. *Ibid.*, I, 159.

—— (1923b). La culture des trypanosomes et les conditions physico-chimiques qui la déterminent. *Ibid.*, I, 181.

PONTANO, T. (1920). Sulla riproduzione dei macrogameti di *Plasmodium vivax* nel circolo sanguigno. *Policlinico*, XXVII, Sez. Med., 36.

—— (1923). Sulla riproduzione sperimentale della malaria per mezzo dei gameti. *Ibid.*, XXX, Sez. Med., 269.

PORTER, A. (1908). A new Schizogregarine, *Merogregarina amaroucii* nov. gen., nov. sp., parasitic in the alimentary canal of the composite ascidian *Amaroucium* sp. (Prel. comm.). *Arch. Zool. Exp.*, IX (N. & R.), xliv.

—— (1908a). *Leucocytozoon musculi* sp. n., a parasitic Protozoön from the blood of white mice. *Proc. Zool. Soc. London*, II, 703.

—— (1909). *Merogregarina amaroucii* nov. gen., nov. sp., a Sporozoön from the digestive tract of the ascidian *Amaroucium* sp. *Arch. Protist.*, XV, 227.

—— (1909a). *Amœba chironomi* n. sp., parasitic in the alimentary tract of the larva of a *Chironomus*. *Parasitology*, II, 32.

—— (1909b). The life-cycle of *Herpetomonas jaculum* (Léger), parasitic in the alimentary tract of *Nepa cinerea*. *Ibid.*, II, 367.

—— (1909c). The morphology and life-history of *Crithidia gerridis*, as found in the British water bug, *Gerris paludum*. *Ibid.*, II, 348.

—— (1910). The structure and life-history of *Crithidia melophagia* (Flu), an endo-parasite of the sheep ked, *Melophagus ovinus*. *Quart. Journ. Micro. Sci.*, LV, 189.

—— (1911). The structure and life-history of *Crithidia pulicis*, n. sp., parasitic in the alimentary tract of the human flea, *Pulex irritans*. *Parasitology*, IV, 237.

—— (1914). The morphology and biology of *Herpetomonas patellæ*, n. sp., parasitic in the limpet, *Patella vulgata*, together with remarks on the pathogenic significance of certain flagellates found in invertebrates. *Ibid.*, VII, 322.

—— (1916). The occurrence of *Pneumocystis carinii* in mice in England. *Ibid.*, VIII, 255.

—— (1918). A survey of the intestinal Entozoa, both protozoal and helminthic, observed among natives in Johannesburg from June to November, 1917. *Pub. S. African Inst. Med. Res.*, No. XI.

POSPIECH, W. (1919). Untersuchungen über den mikroskopischen Nachweis von Darmparasiten im Kot von Fleischfressern, mit besonderer Berücksichtigung der Kokzidien. (Veterinärmed. Inaug. Diss.) Munich.

POUND, C. J., & HUNT (1895). Texas fever in Australia. *12th Ann. Rept. Bur. Anim. Indust.*, U.S. Dept. Agric.. 85.

POWELL, A., & KOHIYAR, A. J. (1920). A *Bodo*-like flagellate persisting in the urinary tract for five years, the urine remaining bacteriologically sterile throughout. *Proc. Roy. Soc. Med.*, XIII, Sect. Path., 1.

PRANDTL, H. (1906). Die Konjugation von *Didinium nasutum* O.F.M. *Arch. Protist.*, VII, 229.

PRESSAT, A. (1905). Le paludisme et les moustiques (prophylaxie). Paris.

PRIESTLY, H. (1915). Theileria tachyglossi (n. sp.), a blood parasite of *Tachyglossus aculeatus*. *Ann. Trop. Med. & Parasit.*, IX, 233.

PRINGAULT, E. (1914). *Cimex pipistrelli* Jen. agent de la transmission de la trypanosomiase des chauves-souris. *C. R. Soc. Biol.*, LXXVI, 881.

—— (1914a). Non-pathogénéité du *Trypanosoma vespertilionis* (Battaglia) pour les animaux de laboratoire. *Ibid.*, LXXVI, 883.

—— (1920). Etude biologique de *Trichomonas intestinalis*. *Bull. Soc. Path. Exot.*, XIII, 800.

PROWAZEK, S. VON (1903). Flagellatenstudien. *Arch. Protist.*, II, 195.

—— (1904). Die Entwicklung von *Herpetomonas*, einem mit den Trypanosomen verwandten Flagellaten. *Arb. K. Gesundheitsamte*, XX, 440.

—— (1904a). Untersuchungen über einige parasitische Flagellaten. *Ibid.*, XXI, 1.

—— (1905). Studien über Säugetiertrypanosomen—I. *Ibid.*, XXII, 351.

—— (1908). Ueber Haemogregarinen aus *Porocephalus moniliformis*. *Zool. Anz.*, XXXIII, 465.

—— (1909). Zysten von *Bodo lacertæ*. Entgegnung zu dem Artikel: " Some remarks upon the ' autogamy' of *Bodo lacertæ* (Grassi)" von C. Dobell d. Zeitschr., Bd. 28, p. 548. *Biol. Centralbl.*, XXIX, 27.

—— (1910). Parasitische Protozoen aus Japan, gesammelt von Herrn Dr. Mine in Fokuoka. *Arch. Schiffs.- u. Trop.-Hyg.*, XIV, 297.

—— (1911). Beitrag zur Entamœba-Frage. *Arch. Protist.*, XXII, 345.

—— (1911a). Zur Kenntnis der Flagellaten des Darmtraktus. *Ibid.*, XXIII, 96.

—— (1912). *Entamœba*. *Ibid.*, XXV, 273.

—— (1912a). Weiterer Beitrag zur Kenntnis der Entamöben. *Ibid.*, XXVI, 241.

—— (1912b). Beiträge zur Kenntnis der Protozoen und verwandter Organismen von Sumatra (Deli)—VII. *Ibid.*, XXVI, 250.

—— (1913). Zur Kenntnis der Balantidiosis. *Arch. Schiffs.- u. Trop.-Hyg.*, XVII (Beih. 6), 369.

—— (1913a). Zur Parasitologie von Westafrika. *Centralbl. Bakt.*, I. Abt., LXX, 32.

—— (1913b). Studien zur Biologie der Protozoen. *Arch. Protist.*, XXXI, 47.

—— (1920. Posthumous). Infusoria-Ciliata. Prowazek's *Handb. d. Path. Prot.*, II. 842.

PROWAZEK, S. VON, & WERNER, H. (1914). Zur Kenntnis der sog. Flagellaten. *Arch. Schiffs.- u. Trop.-Hyg.*, XVIII (Beih. 5), 311.

PULVIRENTI, G. (1911). La leishmaniosi del cane a Catania. *Pathologica*, III, 205.

—— (1913). La leishmaniosi cutanea nelle provincie di Catania e Caltanissetta. *Malaria e Mal. Paesi Caldi*, IV, 32.

PYMAN, F. L., & WENYON, C. M. (1917). The action of certain emetine derivatives on amœbæ. *Journ. Pharm. & Exp. Therap.*, X, 237.

QUINCKE, H., & ROOS, E. (1893). Ueber Amöben-Enteritis. *Berlin. Klin. Woch.*, XXX, 1089.

RABINOWITSCH, L., & KEMPNER, W. (1899). Beitrag zur Kenntnis der Blutparasiten, speciell der Rattentrypanosomen. *Zeitschr. Hyg. u. Infekt.*, XXX, 251.

RAILLIET, A. (1895). *Traité de Zoologie Médicale et Agricole*. 2nd edition. Paris.

RAILLIET, A., & LUCET, A. (1888). Corps oviformes dans les villosités intestinales du chien. *Bull. Soc. Méd. Vét.*, XLII, 370.

—— —— (1890). Observations sur quelques coccidies intestinales. *C. R. Soc. Biol.*, II, 660.

—— —— (1890a). Une nouvelle maladie parasitaire de l'oie domestique, déterminée par des coccidies. *Ibid.*, XLII, 293.

—— —— (1891). Note sur quelques espèces de coccidies encore peu étudiées. *Bull. Soc. Zool.*, Paris, XVI, 246.

—— —— (1891a). Développement expérimentale des coccidies de l'épithélium intestinal du lapin et de la poule. *C. R. Soc. Biol.*, XLIII, 820.

RANGEL, R. (1905). Nota preliminar sobre la peste boba y la desrengadera de los llanos de Venezuela. *Bol. Lab. Hosp. Vargas*, Caracas, II, 11.

RATZ, S. VON (1912). [Über die Piroplasmose der Schafe.] *Allatorvosi Lapok.*, XXXV, 207.

—— (1913). Über die Piroplasmose der Schafe. *Centralbl. Bakt.*, I. Abt., LXVIII, 194.

—— (1913a). Trichomonas aus der Leber der Tauben. *Ibid.*, I. Abt., LXXI, 184.

RAU, A. N. (1925). Haemogregarina canis. *Vet. Journ.*, LXXXI, 293.

RAVAUT, P. (1920). Deux cas de bouton d'orient contractés en Espagne et en France. (Premier cas de contagion en France.) *Bull. Soc. Path. Exot.*, XIII, 235.

RAYER (1846). Œufs de distome (*Distoma lanceolatum*) en quantité innombrable dans les voies biliaires du lapin domestique (*Lepus cuniculus*); sans distome dans les mêmes parties. *Arch. Anat. Gén. & Phys.* Suppl. *Arch. Gén. Méd. Journ. Compl. Sci. Méd.*, 4 Series, 20.

REBAGLIATI, R. (1914). Etiologie de la Uta. *Cronica Medica*, XXXI, 169.

REDI, F. (1708). De animalibus vivis, quæ in corporibus animalium viventium reperiuntur, observationes. Amstelædami, 183.

REES, D. C. (1900). Experimental proof of the malaria-mosquito theory. *Brit. Med. Journ.*, II, 1054.

REICH, F. (1913). Das Kaninchencoccid *Eimeria stiedæ* (Lindemann, 1865) nebst einem Beitrage zur Kenntnis von *Eimeria falciformis* (Eimer, 1873). *Arch. Protist.*, XXVIII, 1.

REICH, W. W. (1924). *Trypanosoma brucei* as a filterable virus. *Journ. Parasit.*, X, 171.

REICH, W. W., & BECKWITH, T. D. (1922). *Trypanosoma brucei* as a filterable virus—a preliminary note. *Ibid.*, IX, 93.

REICHENOW, E. (1910). *Hæmogregarina stepanowi*. Die Entwicklungsgeschichte einer Hämogregarine. *Arch. Protist.*, XX, 251.

—— (1912). Die Hämogregarinen. Prowazek's *Handb. d. Path. Prot.*, II, 602.

—— (1913). *Karyolysus lacertæ*, ein wirtweckselndes Coccidium der Eidechse *Lacerta muralis* und der Milbe *Liponyssus saurarum*. *Arb. K. Gesundheitsamte*, XLV, 317.

—— (1917). Parasitos de la sangre y del intestino de los monos antropomorfos africanos. *Bol. R. Soc. Españ. Hist. Nat.*, XVII, 312.

—— (1917a). Sobre el problema de la immunidad de los negros contra el paludismo. *Bol. Inst. Nac. Hig. Alfonso XIII*, XIII, 29.

—— (1918). *Eutrichomastix lacertæ* en la sangre y en ácaros hématófagos. *Ibid.*, XIV, 183.

—— (1919). Der Entwicklungsgang der Hämococcidien *Karyolysus* und *Schellackia* nov. gen. *Sitzungsb. Ges. Naturf. Freunde*, 440.

—— (1920). Biologische Beobachtungen an Gorilla und Schimpanse. *Ibid.*, 1.

—— (1920a). Den Wiederkäuerinfusorien verwandte Formen aus Gorilla und Schimpanse. *Arch. Protist.*, XLI, 1.

—— (1920b). *Eutrichomastix lacertæ* im Blut und im blutsaugenden Milben. *Centralbl. Bakt.*, I. Abt., LXXXIV, 466.

—— (1920c). Ueber das Vorkommen der Malariaparasiten des Menschen bei den afrikanischen Menschenaffen. *Ibid.*, I. Abt., LXXXV, 207.

—— (1921). Die Hämococcidien der Eidechsen. Vorbemerkungen und I. Theil. Die Entwicklungsgeschichte von *Karyolysus*. *Arch. Protist.*, XLII, 180.

—— (1921a). Die Coccidien. Prowazek's *Handb. d. Path. Prot.*, III, 1136.

—— (1922). Intracelluläre Symbionten bei blutsaugenden Milben und Egeln. *Arch. Protist.*, XLV, 95.

—— (1923). Über Darmflagellatenzüchtung und deren Anwendung zum Nachweis der Flagellaten im Stuhl. *Arch. Schiffs.- u. Trop.-Hyg.*, XXVII, 367.

—— (1925). Über das Vorkommen von zwei Coccidienarten der Gattung *Isospora* beim Menschen. *Ibid.*, XXIX, 172.

REICHENOW, E., & SCHELLACK, C. (1912). Streitfragen in der Coccidienforschung. *Zool. Anz.*, XXXIX, 609.

REINCKE, (1866). Nonnula quædam de psorospermiis cuniculi. (Diss. inaug.) Kiel.

REINECKE (1911). Eine Trypanosomenkrankheit der Dromedare in Deutsche-Sudwest-afrika. *Zeitschr. Vet.*, XXIII, 1.

REIS, VAN DER (1923). Ueber die Bakterienflora des Darms (*Balantidium coli* und pathologische Dünndarmbesiedlung). *Munch. Med. Woch.*, LXX, 835.

REMAK, R. (1845). Diagnostische und pathogenetische Untersuchungen in der Klinik des Herrn Geh. Raths Dr. Schönlein. Berlin, 239.

REULING, F. (1921). Zur Morphologie von *Trichomonas vaginalis* Donné. *Arch. Protist.*, XLII, 347.

REULING, F., & RODENWALDT, E. (1921). *Giardia - Lamblia* ?. *Ibid.*, XLII, 337.

RICHARD, J. (1882). Sur le parasite de la malaria. *Naturaliste*, II, 57.

RICHARDSON, U. F. (1925). [Spleen smears of horse, showing a possible *Leishmania*.] *Trans. Roy. Soc. Trop. Med. & Hyg.*, XIX, 281.

—— (1926). A probable case of equine leishmaniasis. *Ibid.*, XIX, 411.

RICKETTS, H. T. (1906). The transmission of Rocky Mountain spotted fever by the bite of the wood tick (*Dermacentor occidentalis*). *Journ. Amer. Med. Assoc.*, XLVII, 358.

RICKETTS, H. T., & WILDER, R. W. (1910). The ætiology of typhus fever (Tabardillo) of Mexico City. A further preliminary report. *Ibid.*, LIV, 1373.

RIEVEL & BEHRENS (1904). Beiträge zur Kenntnis der Sarcosporidien und deren Enzyme. *Centralbl. Bakt.*, I. Abt., XXXV, 341.

RINGENBACH, J. (1914). Quelques hématozoaires du Congo. *Bull. Soc. Path. Exot.*, VII, 296.

RIVOLTA, S. (1869). Psorospermi e psorospermosi negli animali domestici. *Medico Veter.*, IV.

—— (1873). *Dei parassiti vegetali*. Turin.

—— (1874). *Sopra alcune specie di tenie delle pecore e sopra speciali cellule oviformi dei villi del cane e del gatto*, Pisa, 14.

—— (1877). Delle cellule oviforme dei villi del cane. *Studi Gabi. Anat. Pat. R. Scu. Vet. Pisa*, 42.

—— (1877a). Ancora delle cellule oviformi e specialmente di quelle con nucleo in segmentazioni dei villi del cane. *Ibid.*, 85.

RIVOLTA, S. (1878). Della gregarinosi dei polli e dell' ordinamento delle gregarine e dei psorospermi degli animali domestici. *Giorn. Anat. Fisiol. e Pat. Anim.* Pisa, 1877-78, X, 220; *Studi Gabi. Anat. Pat. R. Scu. Vet.* Pisa, 1879, 57.

RIVOLTA, S., & DELPRATO, P. (1881). *L'ornitoiatria o la medicina degli uccelli domestici e semidomestici.* Pisa.

RIVOLTA, S., & SILVESTRINI, A. (1873). Psorospermi epizootica nei gallinacei. *Giorn. Anat. Fisiol. e Pat. Anim.* Pisa, V, 42.

ROBERTSON, A. (1923). Note on a case infected with *Dientamœba fragilis*, Jepps and Dobell, 1917. *Journ. Trop. Med. & Hyg.*, XXVI, 243.

ROBERTSON, M. (1907). Studies on a trypanosome found in the alimentary canal of *Pontobdella muricata. Proc. R. Phys. Soc. Edin.*, XVII, 83.

—— (1908). A preliminary note on Hæmatozoa from some Ceylon reptiles. *Spolia Zeylanica*, V, 178.

—— (1909). Studies on Ceylon Haematozoa. No. I.—The life-cycle of *Trypanosoma vittatœ. Quart. Journ. Micro. Sci.*, LIII, 665.

—— (1909a). Further notes on a trypanosome found in the alimentary tract of *Pontobdella muricata. Ibid.*, LIV, 119.

—— (1910). Studies on Ceylon Hæmatozoa. No. II.—Notes on the life-cycle of *Hæmogregarina nicoriœ*, Cast. and Willey. *Ibid.*, LV. 741.

—— (1911). Transmission of flagellates living in the blood of certain fresh-water fishes. *Phil. Trans. Roy. Soc.*, B, CCII, 29.

—— (1912). Notes on some flagellate infections found in certain Hemiptera in Uganda. *Proc. Roy. Soc.*, B, LXXXV, 234; *Rep. Sleep. Sickn. Comm. Roy. Soc.*, XII, 132.

—— (1912a). Notes on certain aspects of the development of *Trypanosoma gambiense* in *Glossina palpalis. Proc. Roy. Soc.*. B, LXXXV, 241.

—— (1912b). Notes on the polymorphism of *Trypanosoma gambiense* in the blood, and its relation to the exogenous cycle in *Glossina palpalis. Ibid.*, LXXXV, 527.

—— (1913). Notes on the life-history of *Trypanosoma gambiense*, with a brief reference to the cycles of *Trypanosoma nanum* and *Trypanosoma pecorum* in *Glossina palpalis. Phil. Trans. Roy. Soc.*, B, CCIII, 161.

ROBERTSON, W. (1901). Malignant jaundice in the dog. *Journ. Comp. Path. & Therap.*, XIV, 327.

ROCHA-LIMA, H. DA (1912). Beitrag zur Kenntnis der Blastomycosen. Lymphangitis epizootica und Histoplasmosis. *Centralbl. Bakt.*, I. Abt., LXVII, 233.

—— (1916). Beobachtungen bei Flecktyphusläusen. *Arch. Schiffs.- u. Trop.-Hyg.*, XX, 17.

—— (1916a). Zur Aetiologie des Fleckfiebers. (Vorl. Mitt.) *Berlin. Klin. Woch.*, LIII, i, 567.

ROCHA-LIMA, H. DA, & WERNER, H. (1913). Ueber die Züchtung von Malariaparasiten nach der Methode von Bass. *Arch. Schiffs.- u. Trop.-Hyg.*, XVII, 541.

ROCHE, W. (1917). Intestinal Protozoa in Salonika war area. *Lancet*, I, 297.

RODENHUIS, Y. (1919). Bijdrage tot de kennis van de joodcysten en hunne amœben. *Geneesk. Tijdschr. Nederl.-Indië*, LIX, 813.

RODENWALDT, E. (1912). Flagellaten (*Trichomonas, Lamblia*). Prowazek's *Handb. d. Path. Prot.*, I, 78.

RODHAIN, J. (1913). A propos de *Leptomonas pangoniœ* et *Trypanosoma denysi.* (Note rectificative.) *Bull. Soc. Path. Exot.*, VI, 181.

—— (1915). Quelques hématozoaires de petits mammifères de l'Uele (Ouellé) Congo belge. *Ibid.*, VIII, 726.

—— (1916). Note sur les trypanoses et les piroplasmoses des grands animaux de l'Ouellé. *Ibid.*, IX, 95.

—— (1923). Trypanosome d'un cheiroptère insectivore *Nycteris hispida* Schreber au Congo belge. *Ibid.*, XVI, 659.

RODHAIN, J., & BEQUAERT, J. (1911). Présence de *Leptomonas* dans le latex d'une euphorbe congolaise. *Ibid.*, IV. 198.

—— —— (1916). Histoire de " *Passeromyia heterochœta* " Villen et de " *Stasisia (Cordylobia) rodhaini* " Ged. *Bull. Sci. France & Belgique*, XLIX, 236.

RODHAIN, J., BEQUAERT, J., PONS, C., & VANDENBRANDEN, F. (1911). Notes sur des formes *Leptomonas* constituant une culture d'un trypanosome dans l'intestin de *Pangonia.* Leptomonas d'asilide et de réduviides du Katanga. *Bull. Soc. Path. Exot.*, IV, 528.

—— —— —— —— (1912). Les trypanoses animales au Bas-Katanga et leurs rapports avec les glossines. *Ibid.*, V, 45.

—— —— —— —— (1912a). *Leptomonas pangoniœ* parasite de *Pangonia infusca. Ibid.*, V, 604.

—— —— —— —— (1912b). Essais de transmission du *Trypanosoma gambiense* par la *Glossina morsitans. Ibid.*, V, 762.

—— —— —— —— (1912c). Contribution au mécanisme de la transmission des trypanosomes par les glossines. *Arch. Schiffs.- u. Trop.-Hyg.*, XVI, 732.

—— —— —— —— (1913). Note sur les trypanosomides intestinaux d'Hæmatopota au Congo belge. *Bull. Soc. Path. Exot.*, VI, 182.

—— —— —— —— (1913a). *Rapport sur les travaux de la mission scientifique du Katanga octobre, 1910, à septembre, 1912.* Bruxelles.

ROGERS, L. (1901). The transmission of *Trypanosoma evansi* by horse flies, and other experiments pointing to the probable identity of surra of India and nagana of tsetse-fly disease of Africa. *Proc. Roy. Soc.*, LXVIII, 163.

—— (1904). Preliminary note on the development of *Trypanosoma* in cultures of the Cunningham-Leishman-Donovan bodies of cachexial fever and kala-azar. *Lancet*, II, 215.

—— (1913). *Dysenteries : their differentiation and treatment.* London.

—— (1915). Tartar emetic in kala-azar. *Brit. Med. Journ.*, II, 197.

ROLOFF, F. (1868). Ueber die sogenannten Psorospermien-Knoten in der Leber bei Kaninchen. *Arch. Path. Anat.*, XLIII, 512.

ROOS, E. (1893). Ueber Infusoriendiarrhoe. *Deutsch. Arch. Klin. Med.*, LI, 505.

—— (1894). Zur Kenntnis der Amöbenenteritis. *Arch. Exper. Path. u. Pharm.*, XXXIII, 389.

ROOT, F. M. (1921). Experiments on the carriage of intestinal Protozoa of man by flies. *Amer. Journ. Hyg.*, I, 131.

ROSENBERG, B. (1892). Ein Befund von Psorospermien (Sarkosporidien) im Herzmuskel des Menschen. *Zeitschr. Hyg. u. Infekt.*, XI, 435.

ROSENBUSCH, F. (1909). Trypanosomen Studien. *Arch. Protist.*, XV, 263.

—— (1910). Ueber eine neue Encystierung bei *Crithidia muscœ domesticœ*. *Centralbl. Bakt.*, I. Abt., LIII, 387.

ROSENBUSCH, F., & GONZALEZ, R. (1925). Beitrag zum Studium der *Tristeza*. *Arch. Protist.*, L, 443.

ROSENFELD, A. (1904). Die Bedeutung der Flagellaten im Magen und Darm des Menschen. *Deutsch. Med. Woch.*, XXX, 1717.

ROSS, E. H. (1909). A gregarine parasitic in the dog flea, *Ctenocephalus serraticeps*. *Ann. Trop. Med. & Parasit.*, II, 359.

ROSS, P. H. (1905). A note on the natural occurrence of piroplasmosis in the monkey (*Cercopithecus*). *Journ. Hyg.*, V, 18.

—— (1907). Report on experiments to ascertain the ability of tsetse flies to convey *Trypanosoma gambiense* from infected to clean monkeys and on an intracorpuscular stage of the trypanosome. *Rep. Sleep. Sickn. Comm. Roy. Soc.*, VIII, 80.

—— (1908). MS. report on experiments carried out to ascertain whether other species of *Glossina* besides *G. palpalis* can carry the human trypanosome from infected to non-infected monkeys. *Colon. Rept. East Africa Protec.*, App. II. (Review in *Sleep. Sickn. Bur. Bull.*, I, 213.)

—— (1911). *Nairobi Laboratory Reports for 1904-1910. East Africa Protectorate*, I.

ROSS, R. (1895). The crescent-sphere-flagella metamorphosis of the malaria parasite in the mosquito. *Trans. S. Indian Branch Brit. Med. Assoc.*, VI, 334.

—— (1897). On some peculiar pigmented cells found in two mosquitoes fed on malarial blood. *Brit. Med. Journ.*, II, 1786.

—— (1898). Pigmented cells in mosquitoes. *Ibid.*, I, 550.

—— (1898a). Report on the preliminary investigation into malaria in the Sihur Ghat, Ootacamund. *Trans. S. Indian Branch Brit. Med. Assoc.*, VII, 143.

—— (1898b). Report on the cultivation of *Proteosoma*, Labbé, in grey mosquitoes. Calcutta. [Dated May 21, 1898, issued October, 1898]; reprinted in *Indian Med. Gaz.*, XXXIII, 401, 448.

—— (1899). The cultivation of the malarial quartan parasite in Anopheles. *Brit. Med. Journ.*, II, 608.

—— (1903). [1.] Note on the bodies recently described by Leishman and Donovan. [2.] Further notes on Leishman's bodies. *Ibid.*, II, 1261, 1401.

—— (1906). Notes on the parasites of mosquitoes found in India between 1895 and 1899. *Journ. Hyg.*, VI, 101.

—— (1918). An interim report on the treatment of malaria. Abstract of 2,460 cases. War Office investigations. *Trans. Soc. Trop. Med. & Hyg.*, XI, 179.

ROUBAUD, E. (1907). Transmission de *Trypanosoma dimorphon* par *Glossina palpalis* R. Desv. (Note préliminaire.) *Ann. Inst. Pasteur*, XXI, 466.

—— (1908). Fixation, multiplication, culture d'attente des trypanosomes pathogènes dans la trompe des mouches tsétsé. *C. R. Acad. Sci.*, CXLVI, 423.

—— (1908a). Sur un nouveau flagellé, parasite de l'intestin des muscides, au Congo français. *C. R. Soc. Biol.*, LXIV, 1107.

—— (1908b). *Leptomonas mesnili* n. sp., nouveau flagellé à formes trypanosomes de l'intestin de muscides non piqueurs. *Ibid.*, LXV, 39.

—— (1909). Les trypanosomes pathogènes et la *Glossina palpalis*. *Rap. Miss. d'Etudes. Malad. Sommeil Congo Franç., 1906-1908*, Paris, 511.

—— (1910). Influence des réactions physiologiques des glossines sur le développement salivaire et la virulence des trypanosomes pathogènes. *C. R. Acad. Sci.*, CLI, 729.

—— (1910a). Précisions relatives aux phénomènes morphologiques du développement des trypanosomes chez les glossines. *Ibid.*, CLI, 1156.

—— (1911). *Cystotrypanosoma intestinalis* n. sp.; trypanosome vrai à reproduction kystique, de l'intestin des mouches vertes (Lucilies) de l'Afrique tropicale. *C. R. Soc. Biol.*, LXXI, 306.

—— (1911a). *Cercoplasma* (n. gen.) *caulleryi* (n. sp.); nouveau flagellé à formes trypanosomiennes de l'intestin d'*Auchmeromyia luteola* Fabr. (muscide). *Ibid.*, LXXI, 503.

ROUBAUD, E. (1911b). Sur un type nouveau de leptomonades intestinales des muscides, *Leptomonas soudanensis* n. sp., parasite des pycnosomes africains. *Ibid.*, LXXI, 570.

—— (1911c). Phénomènes autogamiques chez les *Leptomonas* et formes affines; valeur sexuelle autogame des formes trypanosomiennes des leptomonades, et des formes leptomonadiennes des trypanosomes. *Ibid.*, LXXI, 602.

—— (1912). *Cystotrypanosoma grayi* (Novy), trypanosome propre de *Glossina palpalis*. Polymorphisme, affinités, intérêt phylogénétique. *Ibid.*, LXXII, 440.

—— (1912a). Expériences de transmission de flagellés divers chez les muscides africains du genre *Pycnosoma*. *Ibid.*, LXXII, 508.

—— (1912b). Phénomènes autogamiques et formes trypanosomiennes chez quelques flagellés de muscides africains. *Ibid.*, LXXII, 552.

—— (1912c). Sur un nouveau flagellé à forme trypanosome des drosophiles d'Afrique, *Cercoplasma drosophilae* n. sp. *Ibid.*, LXXII, 554.

—— (1913). Evolution comparée des trypanosomes pathogènes chez les glossines. *Bull. Soc. Path. Exot.*, VI, 435.

—— (1918). Recherches sur la transmission du paludisme par les anophèles français de régions non palustres (Yonne et région parisienne). *Ann. Inst. Pasteur*, XXXII, 430.

—— (1919). Un deuxième type d'hémogrégarine humaine. *Bull. Soc. Path. Exot.*, XII, 76.

ROUBAUD, E., & FRANCHINI, G. (1922). Infection naturelle de la souris par les flagellés de la puce *Ctenopsylla musculi* Dug. *Ibid.*, XV, 405.

—— —— (1922a). Infection leishmaniforme produite chez la souris par l'inoculation des fèces de la puce *Ctenopsylla musculi*. *Ibid.*, XV, 407.

—— —— (1923). Culture des parasites leishmaniformes obtenus chez la souris par inoculation des fèces de la puce *Ctenopsylla musculi*. *Ibid.*, XVI, 14.

ROUDSKY, D. (1910). Sur l'inoculation de cultures de *Trypanosoma lewisi* au rat blanc et sur la réceptivité de la souris blanche à ce trypanosome. *C. R. Soc. Biol.*, LXVIII, 421, 458.

—— (1910a). Sur le *Trypanosoma lewisi* Kent renforcé. *Ibid.*, LXIX, 384.

—— (1911). Sur la possibilité de rendre le *Trypanosoma lewisi* virulent pour d'autres rongeurs que le rat. *C. R. Acad. Sci.*, CLII, 56.

—— (1911a). Action pathogène du *Trypanosoma lewisi* Kent renforcé, sur la souris blanche. *C. R. Soc. Biol.*, LXX, 741.

—— (1912). Sur la réceptivité du *Trypanosoma duttoni* Thiroux. *Ibid.*, LXXII, 221.

ROW, R. (1909). The development of the parasite of oriental sore in cultures. *Quart. Journ. Micr. Sci.*, LIII, 747.

—— (1910). Further observations on *Leishmania tropica* of oriental sore of Cambay, India. *Brit. Med. Journ.*, II, 867

—— (1912, 1913). Some experimental facts re kala-azar (Indian). *Journ. Trop. Med. & Hyg.*, XV, 327; XVI, 1.

—— (1914). Evolution of the diagnostic methods in kala-azar, with a special reference to a technique for intensive culture from the patient's finger blood. *Trans. Grant College Med. Soc.*, Bombay.

—— (1914a). Experimental leishmaniasis in the monkey and the mouse induced by the parasites in culture. *Indian Journ. Med. Res.*, I, 617.

—— (1915). On *Leptomonas davidi* (plant Leishmania) from *Euphorbia pilulifera, Euph. thymifolia, Euph. neruri*, etc. *Trans. Grant College Med. Soc.*, Bombay.

—— (1917). On a simplified technique of Bass's method of cultivating malarial parasites *in vitro*, and a few observations on the malarial parasites cultured by this technique. *Indian Journ. Med. Res.*, IV, 388.

—— (1924). On some pathological observations in experimental leishmaniasis in mice, with special reference to generalized infections set up by *Leishmania tropica*. *Ibid.*, XII, 435.

—— (1925). Canine leishmaniasis in Bombay. *Indian Med. Gaz.*, LX, 317.

RUDOLPHI, C. A. (1819). *Entozoorum synopsis cui accedunt mantissa duplex et indices locupletissimi*. Berolini, 197.

RUDOVSKY, F. (1921). Über Rattenamöbiase. *Wien. Tierärztl. Monatsschr.*, VIII, 189.

—— (1923). Beobachtungen an Protozoen. *Ibid.*, X, 142.

RUGE, R. (1901). Untersuchungen über das deutsche *Proteosoma*. *Centralbl. Bakt.*, I. Abt., XXIX, 187.

RUTELLI, G. (1914). Infezione sperimentale della Leishmania umana nel topolino. *Lavori della Soc. Ital. di Path. Esotica*, 4ᵃ riunione tenuta a Messina il 27-28 guigno, Roma, 64.

SACEGHEM, R. VAN (1921). La trypanosomiase du Ruanda. *C. R. Soc. Biol.*, LXXXIV, 283.

SAMBON, L. W., & LOW, G. C. (1901). Report on two experiments on the mosquito-malaria theory. *Trans. Med.-Chir. Soc.*, LXXXIV, 497.

SAMBON, L. W., & SELIGMANN, C. G. (1907). The hæmogregarines of snakes. *Trans. Path. Soc.*, London, LVIII, 310.

SAMPSON, S. E. (1917). Clinical cases—coccidiosis in the horse. *Vet. Record*, XXX, 130.

SAMSONOFF, P. F. (1925). Zur Frage über einige morphologische Besonderheiten der Pathogenese und die Bedeutung derselben bei den späteren Rezidiven der Malaria. *Centralbl. Bakt.*, I. Abt., XCVI, 109.

SANGIORGI, G. (1911). Sulla presenza di forme di Leishmania infantum (Nicolle) nella pulce (*Pulex serraticeps*) dei cani randagi di Cantania. *Pathologica*, III, 23, 89.

—— (1911a). Beitrag zum Studium eines *Coccidiums* (*Klossiella muris*). Kritische und experimentelle Studie. *Centralbl. Bakt.*, I. Abt., LX, 523.

—— (1912). Leucocytogregarina musculi. *Pathologica*, IV, 380; *Giorn. R. Accad. Med. Torino*, LXXV, 121; *Centralbl. Bakt.*. I. Abt., LXVI, 287.

—— (1913). Un nuovo protozoo parassita del *Mus musculus*. *Pathologica*, V, 323.

—— (1914). Leucocytogregarina cuniculi n. sp. *Ibid.*, VI, 49.

—— (1914a). Toxoplasma ratti, n. sp. *Giorn. R. Accad. Med. Torino*. LXXVII, 383.

—— (1916). Di un coccidio parassita del rene della cavia. *Pathologica*, VIII, 49.

—— (1917). Flagellati nell' intestino umano. *Ibid.*, IX, 193.

—— (1919). Balantidiosi e balantidii in Albania. *Ibid.*, XI, 35.

—— (1922). Sulla presunta esistenza del genre " *Hæmotrichomonas.*" *Annali d'Igiene*, XXXII, 941.

—— (1922a). Di un flagellato dell' intestino umano. *Pathologica*. XIV, 69.

—— (1922b). Blastomicosi spontanea nei muridi. *Ibid.*, XIV, 493.

SANGIORGI, G., & UGDULENA, G. (1917). Ciliati nell' intestino umano. *Ibid.*, IX, 1.

SANT' ANNA, F. (1913). Trabalhos experimentaes sobre um caso de leishmaniase de origem Brazileira. *Med. Contemp.*, Lisbon, XXXI, 267.

SAVAGE, R. E., & YOUNG, J. R. (1917). Report on the treatment of fifty-nine cases of *Entamœba histolytica* infection. *Journ. R. Army Med. Corps*, XXIX, 249.

SCHARDINGER, F. (1899). Der Entwicklungskreis einer *Amœba lobosa* (*Gymnamœba*) : *Amœba gruberi*. *Sitzungsb. Akad. Wiss. Wien.*, Math-Nat. Kl., CVIII, I Abt., 713.

SCHAT, P. (1902). Eenige mededeelingen over surra en omtrent onderzoekingen daarmee in verband staande. *Arch. Java Suiker.*, Soerabaia, X, 193.

SCHAUDINN, F. (1895). Ueber den Dimorphismus der Foraminiferen. *Sitzungsb. Ges. Naturf. Freunde*, 87.

—— (1896). Über den Zeugungskreis von *Paramœba eilhardi* n. g., n. sp. *Sitzungsb. Akad. Wiss.*, Berlin, I, 31.

—— (1896a). Über die Kopulation von *Actinophrys sol* Ehrbg. *Ibid.*, I, 83.

—— (1896b). Über das Centralkorn der Heliozoen, ein Beitrag zur Centrosomenfrage. *Verhandl. Deutsch. Zool. Ges.*, 113.

—— (1899). Über den Generationswechsel der Coccidien und neuere Malariaforschung. *Sitzungsb. Ges. Naturf. Freunde*. 159.

—— (1900). Untersuchungen über den Generationswechsel bei Coccidien. *Zool. Jahrb.*, Anat. Abt.. XIII. 197.

—— (1902). Studien über krankheitserregende Protozoen. I. *Cyclospora caryolitica* Schaud., der Erreger der perniciösen Enteritis des Maulwurfs. *Arb. K. Gesundheitsamte*, XVIII, 378.

—— (1902a). Studien über Krankheitserregende Protozoen. II. *Plasmodium vivax* (Grassi & Feletti) der Erreger des Tertianfiebers beim Menschen. *Ibid.*, XIX, 169.

—— (1903). Untersuchungen über die Fortpflanzung einiger Rhizopoden. *Ibid.*, XIX, 547.

—— (1904). Generations- und Wirtswechsel bei *Trypanosoma* und *Spirochæte*. (Vorl. Mitt.) *Ibid.*, XX, 387.

SCHAUDINN, F., & SIEDLECKI, M. (1897). Beiträge zur Kenntnis der Coccidien. *Verhandl. Deutsch. Zool. Ges.*, 192.

SCHEER, A. VAN DER, & BERLEKOM, J. J. B. VAN (1900). Malaria en Muskieten in Zeeland. *Weekbl. Nederl. Tijdschr. Geneesk.*, XXXVI, 537.

SCHEIN, H. (1907). Hématozoaires des bovidés en Indo-Chine. *Ann. Inst. Pasteur*, XXI, 659.

—— (1911). Piroplasmose du cheval dans le Sud Annam. *Bull. Soc. Path. Exot.*, IV, 39.

—— (1917). Piroplasmose du cheval dans le Sud Annam. *Ibid.*, X, 871.

—— (1921). Les piroplasmoses en Indochine. *Bull. Agric. Inst. Scient. Saigon*, III, 269.

—— (1923). Piroplasmoses d'Indochine. *Bull. Soc. Path. Exot.*, XVI, 30.

SCHELLACK, C. (1912). Untersuchungen über die Coccidien aus *Lithobius* und *Scolopendra* (*Barrouxia, Adelea, Eimeria*). *Verhandl. Deutsch. Zool. Ges.*, XXII, 163.

—— (1913). Coccidien-Untersuchungen. II. Die Entwicklung von *Adelina dimidiata* A. Schn., einem Coccidium aus *Scolopendra cingulata* Latr. *Arb. K. Gesundheitsamte*, XLV, 269.

SCHELLACK, C., & REICHENOW, E. (1910). Neue Beiträge zur Kenntnis der *Lithobius*-Coccidien *Zool. Anz.*, XXXVI. 380.

—— —— (1913). Coccidien-Untersuchungen. I. *Barrouxia schneideri*. *Arb. K. Gesundheitsamte*, XLIV, 30.

—— (1915). Coccidien-Untersuchungen. III. *Adelea ovata* A. Schn. *Ibid.*, XLVIII, 425.

SCHELLHASE, W. (1912). Eine Beobachtung über das Vorkommen von Marginalpoints (*Anaplasma marginale*) im Blut von Schafen in Deutsch-Ostafrika. *Berlin. Tierärztl. Woch.*, XXVIII, 511.

SCHELLHASE, W. (1913). Beobachtungen über die Anaplasmosis und Piroplasmosis der Schafe und Ziegen in Deutsch-Ostafrika. *Zeitschr. Infektionskr. d. Haust.*, XIII, 349.

—— (1914). Ein Beitrag zur Kenntnis der Piroplasmosis der Schafe und Esel. Therapeutische Versuche mit Trypanblau. Über die Anaplasmosis der Esel. *Ibid.*, XV, 93.

SCHILLING, C. (1910). Das Vorkommen von Autogamie bei *Trypanosoma lewisi*. *Arch. Protist.*, XIX, 119.

SCHILLING, C., & FRIEDRICH (1912). Über Immunität bei *Pirosoma canis*. *Zeitschr. Imm.-Forsch.*, XIV, 706.

SCHINDERA, M. (1922). Beiträge zur Biologie, Agglomeration und Züchtung von *Trypanoplasma helicis* Leidy. *Arch. Protist.*, XLV, 200.

SCHINGAREFF, A. J. (1906). Des Hémosporidies des chauves-souris. *Arch. Sci. Biol.*, St. Petersburg, XII, 181.

SCHIRCH, P. (1914). Beiträge zur Kenntnis des Lebenscyclus von *Arcella vulgaris* Ehrbg. und *Pelomyxa palustris* Greeff. *Arch. Protist.*, XXXIII, 247.

SCHMIDT, A. (1895). Ueber parasitäre Protozoen (*Trichomonas pulmonalis*) im Auswurf. *Münch. Med. Woch.*, XLII, 1181.

SCHMIDT, R. (1904). Infusorien im Mageninhalt bei *Ulcus ventriculi*. *Wien. Klin. Woch.*, XVII, 1304.

SCHMIDT, W. (1920). Untersuchungen über *Octomitus intestinalis truttæ*. *Arch. Protist.*, XL, 253.

SCHNEIDER. A. (1875). Contributions à l'histoire des grégarines des invertébrés de Paris et de Roscoff. *Arch. Zool. Exp.*, IV, 493.

—— (1875a). Note sur la psorospermie oviforme du poulpe. *Ibid.*, IV (N. & R.), xl.

—— (1881). Sur les psorospermies oviformes ou coccidies, espèces nouvelles ou peu connues. *Ibid.*, IX, 387.

—— (1883). Nouvelles observations sur la sporulation du *Klossia octopiana*. *Ibid.*, I, 77.

—— (1885). *Tablettes zoologiques*. Poitiers, I.

—— (1887). *Tablettes zoologiques*. Poitiers, II.

—— (1892). Le cycle évolutif des coccidies et M. le docteur L. Pfeiffer. *Tablettes Zoologiques*, Poitiers, II, 105.

SCHOENING. H. W. (1924). Trypanosomiasis in camels. Its detection by complement fixation. *Journ. Infect. Dis.*, XXXIV, 608.

SCHOO, H. J. M. (1902). La malaria in Olanda. *Atti Soc. Studi d. Malaria*, Rome, III, 189.

—— (1903). La malaria in Olanda nel 1902. *Ibid.*, IV, 85.

SCHRÖDER, O. (1907). Beiträge zur Entwicklungsgeschichte der Myxosporidien. *Sphæromyxa labrazesi* [*sabrazesi*] (Laveran et Mesnil). *Arch. Protist.*, IX, 359.

—— (1909). *Thelohania chætogastris*, eine neue in *Chætogaster diaphanus* Gruith schmarotzende Microsporidienart. *Ibid.*, XIV, 119.

—— (1910). Über die Anlage der Sporocyste (Pansporoblast) bei *Sphæromyxa sabrazesi* Laveran et Mesnil. *Ibid.*, XL, 27.

SCHUBERG, A. (1888). Die Protozoen des Wiederkäuermagens. I. *Bütschlia, Isotricha, Dasytricha, Entodinium*. *Zool. Jahrb.*, III, 365.

—— (1891). Ueber einige Organizationsverhältnisse der Infusorien des Wiederkäuermagens. *Sitzungsb. Phys.-Med. Ges. Wurzburg*, 122.

—— (1892). Über Coccidien des Mäusedarmes. *Ibid.*, 65.

—— (1895). Die Coccidien aus dem Darme der Maus. *Verhandl. Naturh.-Med. Ver. Heidelberg* (N.F.), V, 369.

—— (1910). Ueber Mikrosporidien aus dem Hoden der Barbe und durch sie verursachte Hypertrophie der Kern. *Arb. K. Gesundheitsamte*, XXXIII, 401.

SCHUBERG, A., & KUHN, PH. (1911). Ueber die Uebertragung von Krankheiten durch einheimische stechende Insekten. I. Teil. *Ibid.*, XXXI, 377.

SCHUBERG, A., & KUNZE, W. (1906). Über eine Coccidienart aus dem Hoden von *Nephelis vulgaris* (*Herpobdella atomaria*), *Orcheobius herpobdellæ* nov. gen., nov. sp. *Verhandl. Deutsch. Zool. Ges.*, XVI, 233.

SCHUBERG, A., & REICHENOW E. (1912). Über Bau und Vermehrung von *Babesia canis* im Blute des Hundes. *Arb. K. Gesundheitsamte*, XXXVIII, 415.

SCHUBOTZ, H. (1905). Beiträge zur Kenntnis der *Amœba blattæ* (Bütschli) und *Amœba proteus* (Pall.). *Arch. Protist.*, VI, 1.

—— (1907). *Pycnothrix monocystoides*, nov. gen., nov. spec., ein neues ciliates Infusor aus dem Darm von *Procavia* (*Hyrax*) *capensis* (Pallas). *Denkschr. Med.-Naturw. Ges.*, Jena, XIII, 1.

SCHULTZ (1899). *Colpoda cucullus* im Darm des Menschen. *Berlin. Klin. Woch.*, XXXVI, 353.

SCHULZ, M. (1924). Die Kernteilung von *Leptomonas fasciculata* nebst einem Vergleich mit der Kernteilung bei *Leishmania donovani*. *Arch. Protist.*, XLIX, 216.

SCHUURMANS STEKHOVEN, J. H., JUN. (1919). Die Sexualität der Myxosporidien. *Ibid.*, XL, 27.

—— (1919a). Die Teilung der Trypanosomen. Von Plimmer u. Bradford. *Ibid.*, XL, 158.

SCHWANN, T. (1839). *Mikroskopische Untersuchungen über die Übereinstimmung in der Struktur und dem Wachstum der Thiere und Pflanzen*. Berlin. Translation, Sydenham Soc., London, 1847, XII.

SCOTT, H. H. (1921). The incidence of intestinal parasites, especially with regard to the Protozoa, amongst symptomless carriers in Jamaica. *Ann. Trop. Med. & Parasit.*, XV, 117.

SEEBER, G. R. (1900). Un nuevo esporozoario parasito del hombre dos casos encontrados en pólipos nasales. (Tesis, Univ. Nac. Buenos Aires.)

SEIDELIN, H. (1912). Notes on some blood parasites in man and mammals. *Ann. Trop. Med. & Parasit.*, V, 501.

—— (1912a). Leishmaniasis and babesiasis in Yucatan. *Ibid.*, VI, 295.

—— (1914). *Klossiella* sp. in the kidney of a guinea-pig. *Ibid.*, VIII, 553.

SEIDELIN, H., & CONNAL. A. (1914). A note upon the occurrence of a *Plasmodium* in the blood of West African monkeys. *Ibid.*, VIII, 81.

SELAN, U., & VITTORIO, A. (1924). Nuovo coccidio nel cavallo (*Eimeria utinensis*). *Clin. Vet.*, Milan, XLVII, 587.

SELENEFF, I. F. (1910). Zur Biologie meines Infusoriums, seine Anwesenheit im Prostatasekret. *Centralbl. Bakt.*, I. Abt., LV, 497.

SELLARDS, A. W., & LEIVA, L. (1923). Investigations concerning the treatment of amœbic dysentery. *Philippine Journ. Sci.* XXII, 1.

—— (1923a). The effect of stasis on the development of amœbic dysentery in the cat. *Ibid.*, XXII, 39.

SELLARDS, A. W., & THEILER, M. (1924). Investigations concerning amœbic dysentery. *Amer. Journ. Trop. Med.*, IV, 309.

SENEVET, G. (1912). Sur la fréquence de la leishmaniose canine à Alger et ses variations saisonnières. *Bull. Soc. Path. Exot.*, V, 39.

SENEVET, G., & WITAS, P. (1922). Evolution spontanée de l'infection à *Hæmoproteus columbæ*, chez un pigeon isolé à partir du premier mois de son existence. *Ibid.*, XV, 787.

SERGENT, ED. (1902). Sur une coccidie nouvelle parasite du caméléon vulgaire. *C. R. Soc. Biol.*, LIV, 1260.

—— (1908). Un cas de réveil d'infection à hémocytozoaires chez un singe. *Bull. Soc. Path. Exot.*, I, 187.

—— (1915). Infections expérimentales de la souris par des cultures de la *Leishmania tropica*. *Ibid.*, VIII, 22.

—— (1921). Sur l'hypothèse de l'évolution des " *Sarcocystis* " du bœuf chez un insecte hématophage hôte définitif. *C. R. Soc. Biol.*, LXXXV, 408.

—— (1921a). Etude morphologique du *Piroplasma* (*Gonderia*) *mutans* du bœuf. *Ann. Inst. Pasteur*, XXXV, 193.

—— (1923). De l'association des différentes piroplasmoses entre elles, à propos du prétendu *Piroplasma annulatum* Dschunkowsky et Luhs, 1904. *Bull. Soc. Path. Exot.*, XVI, 23.

SERGENT, ED., & BÉGUET, M. (1914). De l'immunité dans le paludisme des oiseaux. Les pigeons guéris de l'infection à *Hæmoproteus columbæ* ne sont pas immunisés contre elle. *C. R. Soc. Biol.*, LXXVII, 21.

SERGENT, ED., & DONATIEN, A. (1921). De l'infection latente dans la trypanosomiase des dromadaires (le debab). (XXIIe Note.) *Arch. Inst. Pasteur Afrique du Nord*, I, 179.

—— (1922). Transmission naturelle et expérimentale de la trypanosomiase des dromadaires par les stomoxes. *Ibid.*, II, 291.

SERGENT, ED., DONATIEN, A., & LHÉRITIER, A. (1920). Des étalons guéris cliniquement de dourine peuvent rester des porteurs sains de germes pathogènes. *Bull. Soc. Path. Exot.*, XIII, 515.

SERGENT, ED., DONATIEN, A., PARROT, L., LESTOQUARD, F., PLANTUREUX, E., & ROUGEBIEF, H., (1924). Etudes expérimentales sur les piroplasmoses bovines d'Algérie. *Ann. Inst. Pasteur* XXXVIII, 273.

—— —— —— —— (1924). Les piroplasmoses bovines d'Algérie. *Arch. Inst. Pasteur Algérie*, II, 1.

SERGENT, ED., & ET. (1905). Sur des trypanosomes des chauves-souris. *C. R. Soc. Biol.*, LVIII, 53.

—— (1905a). Sur un culicide nouveau très commun à Biskra (*Grabhamia subtilis*). *Ibid.*, LVIII, 673.

—— (1905b). El debab, trypanosomiase des dromadaires de l'Afrique du Nord. *Ann. Inst. Pasteur*, XIX, 17.

—— (1906). Sur le second hôte de l'*Hæmoproteus* (*Halteridium*) du pigeon. (Note préliminaire.) *C. R. Soc. Biol.*, LXI, 494.

—— (1906a) Etudes sur les trypanosomiases de Berbérie en 1905. *Ann. Inst. Pasteur*, XX, 665.

—— (1907). Etudes sur les hématozoaires d'oiseaux: *Plasmodium relictum*, *Leucocytozoon ziemanni* et *Hæmoproteus noctuæ*, *Hæmoproteus columbæ*, trypanosome de l'hirondelle. Algérie, 1906. *Ibid.*, XXI, 251.

—— (1910). Kala-azar. Existence de la leishmaniose chez les chiens d'Alger. (Première note.) *Bull. Soc. Path. Exot.*, III, 510.

—— (1918). Sur le paludisme des oiseaux dû au *Plasmodium relictum* (vel *Proteosoma*). *Ann. Inst. Pasteur*, XXXII, 382.

SERGENT, ED. & ET., & DONATIEN, A. (1920). Deuxième note sur l'hérédité de l'infection et de l'immunité dans la trypanosomiase des dromadaires. *Bull. Soc. Path. Exot.*, XIII, 525.

SERGENT, ED. & ET., LEMAIRE, G., & SENEVET, G. (1914). Insecte transmetteur et réservoir de virus du clou de Biskra. Hypothèse et expériences préliminaires. *Ibid.*, VII, 577.

—————— (1915). Hypothèse sur le phlébotome " transmetteur " et la tarente " reservoir du virus " du bouton d'orient. *Ann. Inst. Pasteur*, XXIX, 309.

SERGENT, ED. & ET., & LHÉRITIER, A. (1919). Passage de trypanosomes de la mère au fœtus dans le " debab." *Bull. Soc. Path. Exot.*, XII, 177.

—— —— (1919a). Fièvre bilieuse hémoglobinurique du bœuf d'Algérie, maladie distincte des piroplasmes. *Ibid.*, XII, 108.

SERGENT, ED. & ET., LHÉRITIER, A., & LEMAIRE, G. (1912). Transmission de *Leishmania* de chien à chien par piqûres de *Pulex serraticeps*. *Ibid.*, V, 595.

SERGENT, ED. & ET., LOMBARD, & QUILICHINI (1912). La leishmaniose à Alger. Infection simultanée d'un enfant, d'un chien et d'un chat dans la même habitation. *Ibid.*, V, 93.

SERGENT, ED. & ET., & PARROT, L. (1922). Sur une hémogrégarine de l'homme observée en Corse. *Ibid.*, XV, 193.

SERGENT, ED. & ET., PARROT, L., DONATIEN, A., & BÉGUET, M. (1921). Transmission du clou de Biskra par le phlébotome (*Phlebotomus papatasi* Scop.). *C. R. Acad. Sci.*, CLXXIII, 1030.

SERGENT, ED. & ET., & SENEVET, G. (1912). Présence d'*Hæmogregarina canis* in Algeria. *Bull. Soc. Path. Exot.*, V, 16.

SERGENT, ED., PARROT, L., & HILBERT, D. N. (1922). Morphologie de piroplasmes observés en Algérie chez le mouton (*Gonderia ovis*). *Ibid.*, XV, 789.

SERGENT, ET. (1919). Influence du froid sur le développement du *Plasmodium relictum* chez le moustique. *Ibid.*, XII, 174.

—— (1921). Trypanosome isolé du sang d'un *Macacus sinicus*. *Arch. Inst. Pasteur Afrique du Nord.*, I, 57.

SERGENT, ET. & ED. (1910). Sur l'immunité dans le paludisme des oiseaux. Conservation *in vitro* des sporozoites de *Plasmodium relictum*. Immunité relative obtenue par inoculation de ces sporozoites. *C. R. Acad. Sci.*, CLI, 407.

—— (1921). Etude expérimentale du paludisme (paludisme des oiseaux à *Plasmodium relictum*, transmis par *Culex pipiens*). *Arch. Inst. Pasteur Afrique du Nord*, I, 1.

—— (1921a). Formes leishmaniennes et leptomonadiennes chez les punaises de chauves-souris. *C. R. Soc. Biol.*, LXXXV, 413.

—— (1921b). Etude expérimentale du paludisme. Paludisme des oiseaux (*Plasmodium relictum*)—I-V. *Bull. Soc. Path. Exot.*, XIV, 72.

—— (1921c). Avantages de la quininisation préventive démontrés et précisés expérimentalement (paludisme des oiseaux). *Ann. Inst. Pasteur.*, XXXV, 125.

SERGENT, ET., GUEIDON, E., BOUGUET, A., & CATANEI, A. (1924). Existence de la leishmaniose cutanée chez le chien dans une localité du Tell algérien où le bouton d'orient est endémique chez l'homme. *Bull. Soc. Path. Exot.*, XVII, 360.

SERGENT, ET., & HEMPL, H. (1917). Sur l'immunité dans le paludisme des oiseaux (*Proteosoma* vel *Plasmodium relictum* Grassi et Feletti). *Ibid.*, X, 550.

SHARP, C. J. H., & MORRISON. H. (1925). Entamœbæ as an aetiological factor in multiple abscesses. *Brit. Med. Journ.*, I, 205.

SHARP, R. G. (1914). *Diplodinium ecaudatum*, with an account of its neuromotor apparatus. *Univ. California Publ. Zool.*, XIII, 43.

SHEATHER, A. L. (1919). A malarial parasite in the blood of a buffalo. *Agric. Res. Inst. Pusa*, Bull. No. 90.

—— (1919a). A malarial parasite in the blood of a buffalo. *Journ. Comp. Path. & Therap.*, XXXII, 223.

—— (1923). The detection of intestinal Protozoa and mange parasites by a flotation technique *Ibid.*, XXXVI, 266.

—— (1924). Coccidiosis in the guinea-pig. *Ibid.*, XXXVII, 243.

—— (1926). A new species of coccidium of the sheep. *Ibid.* XXXIX, 79.

SHIBAYAMA, G. (1910). On malarial parasites of the orang-outang. *Philippine Journ. Sci.*, B, V, 189.

SHIMURA, S. (1918). A new non-pathogenic tetragenous amœba—I. *Journ. Exp. Med.*, XXVIII, 415.

SHORTT, H. E. (1922). Review of the position of the genus *Hæmocystidium* (Castellani and Willey, 1904), with a description of two new species. *Indian Journ. Med. Res.*, IX, 814.

—— (1922a). Notes on two hæmogregarines of Persian lizards. *Ibid.*, IX, 827.

—— (1923). *Herpetomonas ctenocephali*, Fantham : some observations on its life-history and re-actions to different environments. *Ibid.*, X, 721.

—— (1923a). The pathogenicity of insect flagellates to vertebrates, with special reference to *Herpetomonas ctenocephali*, Fantham. *Ibid.*, X, 908.

—— (1923b). Record of kala-azar research work carried out at the King Edward VII Memorial Pasteur Institute, Schillong, during 1922. *Ibid.*, X, 1150.

SHORTT H. E. (1923c). The recovery of *Herpetomonas donovani* from the urine of a kala-azar patient. *Ibid.*, XI, 319.

—— (1925). Note on *Bodo phlebotomi* (*Herpetomonas phlebotomi*) (Mackie, 1914): a reconsideration of its generic position. *Ibid.*, XIII, 141.

SHORTT, H. E., BARRAUD, P. J., & CRAIGHEAD, A. C. (1926). Note on a massive infection of the pharynx of *Phlebotomus argentipes* with *Herpetomonas donovani*. *Ibid.*, XIII, 441.

SHORTT, H. E., & BRAHMACHARI, U. N. (1925). Further observation on dermal leishmanoid. *Ibid.*, XII, 463.

SHORTT, H. E., & SWAMINATH. C. S. (1924). Note on the infection of a mouse by means of bed bugs, *Cimex hemiptera*, Fabr., fed on the peripheral blood of a case of kala-azar. *Ibid.*, XI, 965.

—— —— (1924a). Note on *Nosema adiei* (Christophers, 1922). *Ibid.*, XII, 181.

—— —— (1925). Experiments to decide whether the bed bug, *Cimex hemiptera*, Fabr., can transmit Indian kala-azar. *Ibid.*, XIII, 143.

SHORTT, H. E., SWAMINATH, C. S., & SEN. R. T. (1923). A second report on the recovery of *Herpetomonas donovani* from the urine of kala-azar cases. *Ibid.*, XI, 667.

SIEBOLD, C. T. VON (1839). Beiträge zur Naturgeschichte der wirbellosen Thiere. Ueber Medusa Cyclops, Loligo, Gregarina und Xenos. *Neueste Schrift. d. Naturf. Ges. in Danzig*, III, ii.

—— (1845). Bericht über die Leistungen in der Naturgeschichte der Würmer, Zoophyten und Protozoen während des Jahres 1843 und 1844. *Arch. Naturgesch.*, XI, ii, 256.

—— (1848). *Lehrbuch der vergleichenden Anatomie der wirbellosen Thiere.* Berlin. Heft I.

SIEDLECKI, M. (1898). Etude cytologique et cycle évolutif de la coccidie de la seiche. *Ann. Inst. Pasteur*, XII, 799.

—— (1898a). Reproduction sexuée et cycle évolutif de la coccidie de la seiche (*Klossia octopiana* Schn.). *C. R. Soc. Biol.*, V, 540.

—— (1899). O rozwoju płciowym gregariny: *Monocystis ascidiæ* R. Lank. (Über die geschlechtliche Vermehrung der *Monocystis acsidiæ* R. Lank.) *Bull. Internat. Acad. Sci. Cracovie*, 515.

—— (1899a). Etude cytologique et cycle évolutif de *Adelea ovata* Schneider. *Ann. Inst. Pasteur*, XIII, 169.

—— (1901). Contribution à l'étude des changements cellulaires provoqués par les grégarines. *Arch. Anat. Micro.*, IV, 87.

—— (1902). Historya rozwoju nowego gatunku kokcydyi: *Caryotropha mesnilii* nob. (Cycle évolutif de la *Caryotropha mesnilii*, coccidie nouvelle des polymnies; note préliminaire.) *Bull. Internat. Acad. Sci. Cracovie*, 561.

—— (1907). O budowie i rozwoju *Caryotropha mesnilii*. (Etude de la structure et du cycle évolutif de *Caryotropha mesnilii*.) *Ibid.*, 453.

SIKORA. H. (1918). Beiträge zur Kenntnis der Rickettsien. *Arch. Schiffs.-u. Trop.-Hyg.*, XII, 442.

SILCOCK, A. Q. (1890). A case of parasiticism by psorospermia. *Trans. Path. Soc.*, London, XLI, 320.

SILVA. PEREIRA DA (1913). Notes sur le kala-azar. *Arq. R. Inst. Bact., Camara Pestana*, IV. 147.

—— (1915). Expériences sur la transmission de la leishmaniose infantile par les puces (*Pulex irritans*). *Ibid.*, IV, 261.

SILVERMAN. D. N. (1923). Diarrhœa and disturbances of digestive functions. *New Orleans Med. & Surg. Journ.*, LXXV, 592; *Journ. Amer. Med. Assoc.*, LXXX, 1487.

SIMON, C. E. (1921). *Giardia enterica*—a parasitic intestinal flagellate of man. *Amer. Journ. Hyg.*, I, 440.

—— (1922). A critique of the supposed rodent origin of human giardiasis. *Ibid.*, II, 406.

SIMON, S. K. (1922). Further observations of *Lamblia intestinalis* infestation and its treatment. *Southern Med. Journ.*, XV, 458.

SIMOND, P. L. (1897). L'évolution des sporozoaires du genre *Coccidium*. *Ann. Inst. Pasteur*, XI, 545.

—— (1901). Sur un hématozoaire endoglobulaire pigmentée des tortues. *C. R. Soc. Biol.*, LIII, 150.

—— (1901a). Contribution à l'étude des hématozoaires endoglobulaires des reptiles. *Ann. Inst. Pasteur*, XV, 319.

—— (1903). Note sur un sporozoaire du genre *Nosema*, parasite du *Stegomyia fasciata*. *C. R. Soc. Biol.*, LV. 1335.

SIMONS, H. (1920). Eine saprophytische Oscillarie im Darm des Meerschweinchens. *Centralbl. Bakt.*, II. Abt., L, 356.

—— (1921). Ueber *Selenomonas palpitans* n. sp. *Ibid.*, I. Abt.. LXXXVII, 50.

SIMPSON, J. J. (1918). Bionomics of tsetse and other parasitological notes in the Gold Coast. *Bull. Entom. Res.*, VIII, 193.

SINGH, K. (1925). A further note on Surra transmission experiments with ticks. *Dept. Agric. Punjab,* Vet. Bull. 16.

SINTON, J. A. (1912). Some observations on the morphology and biology of *Prowazekia urinaria* (*Bodo urinarius* Hassall). *Ann. Trop. Med. & Parasit.*, VI, 245.

SINTON, J. A. (1922). A simplified method for the cultivation of *Plasmodium falciparum in vitro*. *Indian Journ. Med. Res.*, X, 203.

—— (1922a). Further remarks on a clinical method for the cultivation of the subtertian malarial parasite *in vitro*, with a note on its use in diagnosis. *Ibid.*, X, 210.

—— (1922b). A case of malaria due to *Plasmodium tenue* (Stephens). *Indian Journ. Med. Res.*, X, 215.

—— (1922c). The situation of the malarial parasite in relation to the red blood-corpuscle. *Indian Med. Gaz.*, LVII, 367.

—— (1925). Bodies resembling hæmogregarines found in smears of human blood. *Indian Journ. Med. Res.*, XIII, 29.

SIVORI, F., & LECLER, E. (1902). Le surra américain ou mal de caderas. *Anal. Min. Agric. Buenos Aires*, V, 1.

SJÖBRING, N. (1897). Beiträge zur Kenntnis einiger Protozoen. *Centralbl. Bakt.*, I. Abt., XXII, 675.

SLUITER, C. P., SWELLENGREBEL, N. H., & IHLE, J. E. W. (1922). De dierlijke parasieten van den mensch en van onze huisdieren. 3rd. edit., Amsterdam, 121.

SMALLMAN, A. B. (1913). Note on some cellular bodies found in a case of Mediterranean leishmaniasis. *Journ. R. Army Med. Corps*, XXI, 636.

SMITH, A. J., & BARRETT, M. T. (1915). The parasite of oral endamebiasis, *Endameba gingivalis* (Gros). *Journ. Parasit.*, I, 159.

SMITH, A. J., MIDDLETON, W. S., & BARRETT. M. T. (1914). The tonsils as a habitat of oral endamebas. Possibility of systemic complications of oral endamebiasis. *Journ. Amer. Med. Assoc.*, LXIII, 1746.

SMITH, S. (1924). Amœbiasis in Secunderabad. *Journ. R. Army Med. Corps*, XLII, 438.

SMITH. T. (1889). Some observations on coccidia in the renal epithelium of the mouse. *Journ. Comp. Med. & Surg.*, X, 211.

—— (1895). An infectious disease among turkeys caused by Protozoa (infectious entero-hepatitis). *U.S. Dept. Agric. Bur. Anim. Indust.*, Bull. No. 8, 7.

—— (1901). The production of sarcosporidiosis in the mouse by feeding infected muscular tissue. *Journ. Exp. Med.*, VI, 1.

—— (1905). Further observations on the transmission of *Sarcocystis muris* by feeding. *Journ. Med. Res.*, XIII, 429.

—— (1910). Intestinal amœbiasis in the domestic pig. *Ibid.*, XXIII, 423.

SMITH, T., & FLORENCE, L. (1925). *Encephalitozoon cuniculi* as a kidney parasite in the rabbit. *Journ. Exp. Med.*, XLI. 25.

SMITH, T., & GRAYBILL, H. W. (1918). Coccidiosis in young calves. *Ibid.*, XXVIII, 89.

—— (1920). Blackhead in chickens and its experimental production by feeding embryonated eggs of *Heterakis papillosa*. *Ibid.*, XXXII, 143.

SMITH, T., & JOHNSON, H. P. (1902). On a *Coccidium* (*Klossiella muris* gen. et sp. nov.) parasitic in the renal epithelium of the mouse. *Ibid.*, VI, 303.

SMITH, T., & KILBORNE, F. E. (1893). Investigations into the nature, causation, and prevention of Texas or southern cattle fever. *U.S. Dept. Agric., Bur. Anim. Indust.*, Bull. 1. 177.

SMITH, T., & SMILLIE, E. W. (1917). Note on coccidia in sparrows and their assumed relation to blackhead in turkeys. *Journ. Exp. Med.*, XXV, 415.

SMITS, J. C. (1916). Mededeelingen uit het Centraal-Hospitaal der Sennah Rubber Company Ltd. to Bila (oostkust van Sumatra). IV. Klinische aanteekeningen op de jaarrapporten over 1914. Malaria, dysenterie, kala-azar, beri-beri, pellagra. *Geneesk. Tijdschr. Nederl.-Indie*, LVI, 138.

SMYLY, H. J., & YOUNG, C. W. (1924). The experimental transmission of leishmaniasis to animals *Proc. Soc. Exp. Biol. & Med.*, XXI, 354.

SNIJDERS, E. P. (1921). On the cysts of a hitherto undescribed species of *Eimeria* in human stools. *Parasitology*, XII, 427.

SOHNS, J. C. F. (1918). *Microbabesia divergens* in Nederlandsch-Indië. *Nederl.-Ind. Bl. v. Diergeneesk. e. Dierent.*, XXX, 385.

SOULIÉ, H., & ROIG, G. (1908). Sur une piroplasmose bacilliforme observée sur les bovins des environs d'Alger. *C. R. Acad. Sci.*, CXLVI, 148.

—— —— (1908a). Piroplasmose bacilliforme bovine observée dans les environs d'Alger. *Ibid.*, CXLVI, 192.

—— —— (1909). Piroplasmose bovine des environs d'Alger. *Ibid.*, CXLVIII, 952.

SPARAPANI, G. C. (1917). Transmissione dell' infezione da *Piroplasma ovis* in tre suini per via digerente. *Pathologica*, IX, 21.

SPIEGL, A. (1919). Zum Vorkommen der Kokzidiose bei Ziegen nebst einigen Bemerkungen über die Biologie der Kokzidien und die Bekämpfung der Kokzidiosen. *Deutsch. Tierärztl. Woch.*, XXVII, 451.

—— (1921). Nieren-Kokzidiose bei Hausgänsen. *Zeitschr. Infektionskr. d. Haust.*, XXII, 263.

—— (1925). Ein bisher nicht bekanntes Kokzid beim Schaf. *Zeitschr. Infektskr. d. Haust.*, XXVIII, 42.

SPLENDORE, A. (1908, 1909, 1910). Un nuovo protozoa parassita dei conigli incontrato nelle lesioni anatomiche d'una malattia che ricorda in molti punti il kala-azar dell' uomo. *Rev. Soc. Scient. S. Paulo*, III, 109; IV, 462; V, 167.

—— (1911). Buba-blastomicosi-leishmaniosi. Nota sopra alcune affezioni framboesiche osservate in Brasile. *Policlinico*, XVIII, 30; *Arch. Schiffs.-u. Trop.-Hyg.*, XV, 105.

—— (1918). Studi nell' interesse di una lotta biologica contro le arvicole. *Boll. Ser. B. Min. Agric. Roma*, 10.

— (1920). Sui parassiti delle arvicole. *Annali d'Igiene*, XXX, 445, 560, 622.

SPREULL, J. (1914). East Coast fever inoculation in the Transkeian territories, S. Africa. *Journ. Comp. Path. & Therap.*, XXVII, 299.

STARCOVICI, C. (1893). Bemerkungen über den durch Babes entdeckten Blutparasiten und die durch denselben hervorgebrachten Krankheiten, die seuchenhafte Hämoglobinurie des Rindes (Babes), das Texasfieber (Th. Smith) und der Carceag der Schafe (Babes). *Centralbl. Bakt.*, I. Abt., XIV. 1.

STATHAM, J. C. B., & BUTLER, G. G. (1913). Note on certain bodies found by liver puncture in a case of fever associated with splenic enlargement. *Journ. R. Army Med. Corps*, XXI, 629.

STEBBINS, J. (1905). On the occurrence of a large-sized parasite of the *Karyolysus* order in the blood of *Rana clamata*. *Centralbl. Bakt.*, I. Abt., XXXVIII, 315.

STEEL, J. H. (1885). *Investigation into an obscure and fatal disease among transport mules in British Burma.*

STEIN, F. (1848). Ueber die Natur der Gregarinen. *Arch. Anat. Phys. Wiss. Med.*, 182.

—— (1852). Neue Beiträge zur Kenntnis der Entwicklungsgeschichte und des feineren Baues der Infusionsthiere. *Zeitschr. Wiss. Zool.*. III. 475.

—— (1854). *Die Infusionsthiere auf ihre Entwicklung untersucht.* Leipzig.

—— (1858). Ueber mehrere neue im Pansen der Wiederkäuer lebende Infusionsthiere. *Abh. K. Böhm. Ges. Wiss.*, X, 69.

—— (1859). Charakteristik neuer Infusoriengattungen. *Lotos*, Prague, IX, 2.

—— (1860). Vortrag über die bisher unbekannt gebliebene *Leucophrys patula* Ehbg. und über zwei neue Infusoriengattungen *Gyrocorys* und *Lophomonas*. *Sitz. K. Böhm. Ges. Wiss.*, I, 44.

—— (1863). Ueber *Paramœcium*(?) *coli* Malmst. *Amtl. Ber.* 37. *Vers. Deutsch. Naturf. u. Ärtze*. 165.

—— (1867-1883). *Der Organismus der Infusionstiere.* II. & III. Abt., Leipzig.

STEINBERG, S. (1862). [An investigation of the soft white matter which accumulates on the teeth and between them.] (In Russian.) *Sovremennaya Meditsina*, Kiev, Nos. 20 and 24, 377, 452.

STEMPELL, W. (1909). Ueber *Nosema bombycis* Nägeli nebst Bemerkungen über Mikrophotographie mit gewöhnlichem und ultraviolettem Licht. *Arch. Protist.*, XVI. 281.

—— (1921). Haplosporidienstudien. I. Neue und wenig bekannte Parasiten aus *Herpetocypris strigata* O. F. Müll. *Ibid.*, XLII. 307.

—— (1921a). Haplosporidinestudien. II. Über *Bertramia beauchampi* n. sp. aus *Conochilus volvox* Ehrbg. *Ibid.*, XLIII, 355.

STEPHENS, J. W. W. (1914). A new malaria parasite of man. *Proc. Roy. Soc.*, B, LXXXVII, 375.

—— (1922). A new malaria parasite of man. *Ann. Trop. Med. & Parasit.*, XVI, 383.

STEPHENS, J. W. W., & BLACKLOCK, B. (1913). On the non-identity of *Trypanosoma brucei* Plimmer & Bradford, 1899, with the trypanosome of the same name from the Uganda ox. *Proc. Roy. Soc.*, B, LXXXVI, 187.

STEPHENS, J. W. W., & CHRISTOPHERS, S. R. (1900). The malarial and blackwater fevers of British Central Africa. *Repts. Malar. Cttee. Roy. Soc.*, Series 1, 12.

—— —— (1900a). Distribution of anopheles in Sierra Leone. *Ibid.*, Series 1, 42.

—— —— (1900b). Note on certain bodies found in the glands of two species of culex. *Further Repts. Cttee. Roy. Soc.*, Series 2, 20.

—— —— (1902). The relation of malarial endemicity to " species " of anopheles. *Repts. Malar. Cttee. Roy. Soc.*, Series 6, 3.

—— —— (1902a). The relation of species of anopheles to malarial endemicity (further report). *Ibid.*, Series 7, 20.

—— —— (1903). Malaria in an Indian cantonment (Mian Mir) and brief summary of conclusions arrived at in the previous papers. *Ibid.*, Series 8, 13.

—— —— (1903a). Note on the changes in the red cell produced by the malignant tertian parasite. *Brit. Med. Journ.*, I, 730.

—— —— (1903b). *The practical study of malaria and other blood parasites.* 1st ed. Lond.

—— —— (1904). *The practical study of malaria and other blood parasites.* 2nd ed. Lond.

—— —— (1908). *The practical study of malaria and other blood parasites.* 3rd ed. Liverpl.

STEPHENS, J. W. W., & FANTHAM, H. B. (1910). On the peculiar morphology of a trypanosome from a case of sleeping sickness and the possibility of its being a new species (*T. rhodesiense*). *Proc. Roy. Soc.*, B, LXXXIII, 28; *Ann. Trop. Med. & Parasit.*, IV, 343.

STEPHENS, J. W. W., YORKE, W., BLACKLOCK, B., MACFIE, J. W. S., FORSTER COOPER, C., & CARTER, H. F. (1918). Studies in the treatment of malaria. VI. Oral administration of quinine for two consecutive days only in simple tertian malaria. *Ann. Trop. Med. & Parasit.*, XI, 283.

STERN, C. (1924). Untersuchungen über Acanthocystideen. *Arch. Protist.*, XLVIII, 436.

STEVENSON, A. C. (1911). Coccidiosis of the intestine in the goat. *4th Rept., Wellcome Trop. Res. Labs., Khartoum*, A, 355.

—— (1915). *Klossiella muris. Quart. Journ. Micro. Sci.*, LXI, 127.

—— (1917). Trypanosomes in the brain tissue of an experimental guinea-pig. *Trans. Soc. Trop. Med. & Hyg.*, XI, 104.

—— (1918). The presence of trypanosomes in brain substance. *Journ. Trop. Med. & Hyg.*, XXI, 17.

—— (1919). [Presence of trypanosomes in the young of infected animals.] *Ibid.*, XXII, 212.

—— (1922). Demonstration of sections showing *Trypanosoma gambiense* in the brain substance of a case of sleeping sickness. *Trans. Roy. Soc. Trop. Med. & Hyg.*, XVI, 135.

STEVENSON, A. C., & WENYON, C. M. (1915). Note on the occurrence of *Lankesteria culicis* in West Africa. *Journ. Trop. Med. & Hyg.*, XVIII, 196.

STIEDA, L. (1865). Ueber die Psorospermien der Kaninchenleber und ihre Entwicklung. *Arch. Path. Anat.*, XXXII, 132.

STILES, C. W. (1891). Note préliminaire sur quelques parasites. *Bull. Soc. Zool.*, Paris, XVI, 163.

—— (1892). Notes on parasites. *Journ. Comp. Med. & Vet. Arch.*, New York, XIII, 319, 517.

—— (1902). The type-species of certain genera of parasitic flagellates, particularly Grassi's genera of 1879 and 1881. *Zool. Anz.*, XXV, 689.

—— (1905). A zoological investigation into the cause, transmission and source of Rocky Mountain "spotted fever." Bull. No. 20, Hyg. Lab., Washington.

STIRLING, R. F. (1921). Existence of *Trypanosoma dimorphon* in Central Provinces of India. *Vet. Record.* I (New Series), 71.

STOCKMAN, S. (1908). Redwater in England and its carriers. *Vet. Journ.*, LXIV, 538.

STOCKMAN, S., & WRAGG, W. G. (1914). Cross-immunization with *Piroplasma bigeminum* and *Piroplasma divergens. Journ. Comp. Path. & Therap.*, XXVII, 151.

STOKES, A. (1888). A preliminary contribution towards a history of the fresh-water Infusoria of the United States. *Journ. Trenton Nat. Hist. Soc.*, I, 71.

STOKVIS, B. J. (1884). Paramœcium in sputa. *Nederl. Tijdschr. Geneesk.*, XX, 4.

ŠTOLC, A. (1899). Actinomyxidia, nová skupina mesozoů, příbuzná *Myxosporidium. Rozpr. Ceske Ak. Cis. Frant. Jos.*, VIII. French résumé, *Bull. Internat. Acad. Sci.*, Bohemia (1901), VI, 18.

STRADA, F., & TRAINA, R. (1900). Ueber eine neue Form von infektiöser Lungenkrankheit der Meerschweinchen. *Centralbl. Bakt.*, I. Abt., XXVIII, 635.

STRICKLAND, C. (1912). *Agrippina bona* nov. gen. et sp. representing a new family of gregarines. *Parasitology*, V, 97.

STRICKLAND, E. H. (1913). Further observations on the parasites of Simulium larvæ. *Journ. Morph.*, XXIV, 43.

STRONG, R. P. (1904). The clinical and pathological significance of *Balantidium coli. Bur. Govt. Lab.*, Manila, Bull. No. 26.

—— (1924). Investigations upon flagellate infections. *Amer. Journ. Trop. Med.*, IV, 345.

STRONG, R. P., TYZZER, E. E., BRUES, C. T., SELLARDS, A. W., & GASTIABURU, J. C. (1915). *Report of First Expedition to South America*, 1913. Harvard School of Tropical Medicine.

STRUBE, G. (1898). *Trichomonas hominis* im Mageninhalt bei Carcinoma cardiæ. *Berlin. Klin. Woch.*, XXXV, 708.

STUHLMANN, F. (1907). Beiträge zur Kenntnis der Tsetsefliege (*Glossina fusca* und *Gl. tachinoides*). *Arb. K. Gesundheitsamte*, XXVI, 301.

SULDEY, E. W. (1924). Dysenterie amibienne spontanée chez le chimpanzé (*Troglodytes niger*). *Bull. Soc. Path. Exot.*, XVII, 771.

SWELLENGREBEL, N. H. (1914). Dierlijke entamoeben uit Deli. *Geneesk. Tijdschr. Nederl.-Indie*, LIV, 420.

—— (1914a). Zur Kenntnis der Entwicklungsgeschichte von *Isospora bigemina* (Stiles). *Arch. Protist.*, XXXII, 379.

SWELLENGREBEL, N. H., SCHÜFFNER, W., & SWELLENGREBEL-DE GRAAF, J. M. H. (1919). The susceptibility of anophelines to malarial infections in Netherlands India. *Meded. Burgerlijk. Geneesk. Dienst. Ned.-Indie*, III, 1.

SWELLENGREBEL, N. H., & STRICKLAND, C. (1910). The development of " *Trypanosoma lewisi* " outside the vertebrate host. *Parasitology*, III, 360.

SWELLENGREBEL, N. H., & SWELLENGREBEL DE GRAAF, J. M. H. (1920). Studies on the various types of malarial infection and the effect of quinine treatment thereon among the native population of the Malay Archipelago. *Ann. Trop. Med. & Parasit.*, XIV, 41.

—— —— (1920a). A malaria survey in the Malay Archipelago. *Parasitology*, XII, 180

SWELLENGREBEL, N. H., & WINOTO, R. M. M. (1917). The life-history of amœbæ of the *limax* type in the human intestine. *Ibid.*, IX, 266.

SWEZY, O. (1915). Binary and multiple fission in *Hexamitus. Univ. California Publ. Zool.*, XVI, 71.

SWEZY, O. (1915a). On a new trichomonad flagellate, *Trichomitus parvus*, from the intestine of amphibians. *Ibid.*, XVI, 89.
—— (1916). The genera *Monocercomonas* and *Polymastix*. *Ibid.*, XVI, 127.
—— (1916a). The kinetonucleus of flagellates and the binuclear theory of Hartmann. *Ibid.*, XVI. 185.
—— (1922). Mitosis in the encysted stages of *Endamœba coli* (Loesch). *Ibid.*, XX, 313.
SWINGLE, L. D. (1909). A study of the life-history of a flagellate (*Crithidia melophagi* n. sp.) in the alimentary tract of the sheep tick (*Melophagus ovinus*). *Journ. Infect. Dis.*, VI, 98.
—— (1911). The transmission of *Trypanosoma lewisi* by rat fleas (*Ceratophyllus* sp. and *Pulex* sp.), with short descriptions of three new herpetomonads. *Ibid.*, VIII, 125.
—— (1911a). The relation of *Crithidia melophagia* to the sheep's blood, with remarks upon the controversy between Dr. Porter and Dr. Woodcock. *Trans. Amer. Micro. Soc.*, XXX, 275.
SYMONS, T. H., & PATTON, W. S. (1912). Report on an outbreak of canine piroplasmosis due to *Piroplasma gibsoni* (Patton) among the hounds of the Madras hunt, together with some observations on the treatment of the disease with salvarsan. *Ann. Trop. Med. & Parasit.*, VI, 361.

TALIAFERRO, W. H. (1923, 1924). A reaction product in infections with *Trypanosoma lewisi* which inhibits the reproduction of the trypanosomes. *Amer. Journ. Hyg.*, III, 204; *Journ. Exp. Med.*, XXXIX, 171.
TALIAFERRO, W. H., & BECKER, E. R. (1922). The human intestinal amoeba, *Iodamoeba williamsi*, and its cysts (iodine cysts). *Amer. Journ. Hyg.*, II, 188.
—— —— (1922a). Society Proceedings, Helminthological Society of Washington, 54th meeting, October 21, 1921. *Journ. Parasit.*, IX, 37.
—— —— (1924). A note on the human intestinal amoeba, *Dientamoeba fragilis*. *Amer. Journ. Hyg.*, IV, 71.
TANABE. M. (1924). Studies on the hæmoflagellata of the loach, *Misgurnus anguillicaudatus*. *Kitasato Arch.*, VI, 121.
—— (1926). Morphological studies in Trichomonas. *Journ. Parasit.*, XII, 120.
TARTAKOWSKY (1905). [Remarks on Dschunkowsky and Luhs' observations on piroplasmosis of cattle in Transcaucasia.] *Proc. VIII. Internat. Congr. Vet. Med.*, Budapest, III, 290.
TAUTE, M. (1911). Experimentelle Studien über die Beziehungen der *Glossina morsitans* zur Schlafkrankheit. *Zeitschr. Hyg. u. Infekt.*, LXIX, 553.
—— (1912). Experimentelle Studien über die Beziehungen der *Glossina morsitans* zur Schlafkrankheit. *Ibid.*, LXXII, 316.
—— (1913). Untersuchungen über die Bedeutung des Grosswildes und der Haustiere für die Verbreitung der Schlafkrankheit. *Arb. K. Gesundheitsamte*, XLV, 102.
TAUTE, M., & HUBER, F. (1919). Die Unterscheidung des "*Trypanosoma rhodesiense*" vom "*Trypanosoma brucei.*" Beobachtungen und Experimente aus dem Kriege in Ostafrika. *Arch. Schiffs.- u. Trop.-Hyg.*, XXIII, 211.
TAYLOR, C. V. (1920). Demonstration of the function of the neuromotor apparatus in *Euplotes* by the method of microdissection. *Univ. California Publ. Zool.*, XIX, 403.
TEICHMANN. E. (1910). Über das Gift der Sarkosporidien. *Arch. Protist.*, XX, 97.
—— (1911). Über die Teilungen der Keime in der Cyste von *Sarcocystis tenella*. *Ibid.*, XXII, 239.
TEICHMANN, E., & BRAUN, H. (1911). Über ein Protozoentoxin (Sarkosporidiotoxin). *Ibid.*, XXII, 351.
TEJERA, E. (1918). [Flagellates of Euphorbias in Venezuela.] *Public. Acad. Méd. Caracas.* (Quoted by Mesnil, 1921.)
—— (1919). *La leishmaniosis americana en Venezuela.* Caracas.
—— (1919a). La trypanosomose américaine ou maladie de Chagas au Vénézuéla. *Bull. Soc. Path. Exot.*, XII, 509.
—— (1919b). The tripanosomosis americana or Chagas's disease in Venezuela. The first case of tripanosomosis americana in the State of Miranda. *Anal. Direc. San.*, Caracas, I, 97, 108.
—— (1920). La leishmaniose américaine au Venezuela. *Bull. Soc. Path. Exot.*, XIII, 238.
—— (1920a). Trypanosomiases animales au Venezuela. *Ibid.*, XIII, 297.
—— (1920b). Un nouveau flagellé de *Rhodnius prolixus*, *Trypanosoma* (ou *Crithidia*) *rangeli* n. sp. *Ibid.*, XIII, 527.
TERRY, B. T. (1911). Trypanosomiasis in monkeys (*Macacus rhesus*) in captivity. *Proc. Soc. Exp. Biol. & Med.*, IX, 17.
—— (1915). The influence that serum exerts upon trypanosomes, with special reference to its use for experiments *in vitro* with atoxyl and paraminophenylarsenoxyd. *Journ. Exp. Med.*, XXI, 250.
THEILER, A. (1903). A new trypanosome and the disease caused by it. *Journ. Comp. Path. & Therap.*, XVI, 193.
—— (1904). Rhodesian tick fever. *Rept. S. Afric. Soc. Advanc. Sci.*, Johannesburg, 201.

THEILER, A. (1904a). The *Piroplasma bigeminum* of the immune ox. *Journ. R. Army Med. Corps*, III, 469.

—— (1904b). East Coast fever. *Ibid.*, III, 599.

—— (1905). Experimentelle Übertragung der tropischen Piroplasmosis des Rindes mittels Zecken. *Fortschr. Vet.-Hyg.*, II, 257.

—— (1905a). Maladies des troupeaux dans l'Afrique du sud. II. Maladies causées par des protozoaires. *Bull. Inst. Pasteur*, III, 657.

—— (1906). Transmission of equine piroplasmosis by ticks in South Africa. *Journ. Comp. Path. & Therap.*, XIX, 283.

—— (1906a). *Piroplasma mutans* (n. spec.) of South African cattle. *Ibid.*, XIX, 292.

—— (1906b). Trypanosomiasis in camels. *Ann. Rept. Direc. Agric. Transvaal, 1904-05*, 106.

—— (1907). Further notes on *Piroplasma mutans*—a new species of piroplasma in South African cattle. Part II. *Journ. Comp. Path. & Therap.*, XX, 1.

—— (1907a). [1.] Inoculation against equine piroplasmosis. [2.] Piroplasmosis in horses due to hyperimmunization. *Rept. Govt. Vet. Bact., Dept. Agric. Transvaal, 1905-06*, 90. 117.

—— (1908). Experiments with English and South African redwater. *Journ. Trop. Vet. Sci.*, IV, 39.

—— (1909). Transmission des spirilles et des piroplasmes par différentes espèces de tiques. *Bull. Soc. Path. Exot.*, II, 293.

—— (1910). *Anaplasma marginale* (gen. and spec. nov.). The marginal points in the blood of cattle suffering from a specific disease. *Rept. Govt. Vet. Bact., Dept. Agric. Transvaal, 1908-09*, 1.

—— (1910a). *Anaplasma marginale* (genus nov. et spec. nov.). Un nouveau protozoaire du bétail. *Bull. Soc. Path. Exot.*, III, 135.

—— (1911). The artificial transmission of East Coast fever. *Rept. Govt. Vet. Bact., Dept. Agric. Transvaal, 1909-10*, 7.

—— (1911a). Progress report on the possibility of vaccinating cattle against East Coast fever. *Rept. Direc. Vet. Res.*, Pretoria, 46.

—— (1912). Weitere Untersuchungen über die Anaplasmosis der Rinder und deren Schutzimpfung. *Zeitschr. Infektionskr. d. Haust.*, XI, 193.

—— (1912a). Übertragung der Anaplasmosis mittels Zecken. *Ibid.*, XII, 105.

—— (1912b). The immunization of cattle against East Coast fever. *Rept. Direc. Vet. Res.*, Pretoria, 266.

THÉLOHAN, P. (1890). Sur deux espèces nouvelles de coccidies parasites de l'épinoche et de la sardine. *C. R. Acad. Sci.*, CX, 1214.

—— (1892). Sur quelques coccidies nouvelles parasites des poissons. *Journ. Anat. & Phys.*, Paris, XXVIII, 151.

—— (1894). Nouvelles recherches sur les coccidies. *Arch. Zool. Exp.*, II, 541.

—— (1895). Recherches sur les myxosporidies. *Bull. Sci. France & Belgique*, XXVI, 100.

THEOBALD, F. V. (1900). A new anopheles (*A. paludis*) from Sierra Leone. *Repts. Malar. Cttee. Roy. Soc.*, Series 1, 75.

THIEL, P. H. VAN (1922). Anopheles en malaria in Leiden en naaste omgeving. (M. D. thesis.) Leiden.

THIROUX (1905). Recherches morphologiques et expérimentales sur *Trypanosoma paddœ* (Laveran et Mesnil). *Ann. Inst. Pasteur*, XIX, 65.

THIROUX, A., & TEPPAZ, L. (1910). Traitement des trypanosomiases chez les chevaux par l'orpiment seul ou associé à l'atoxyl ou à l'émétique de potasse. Traitement du surra chez le dromadaire par l'orpiment seul ou associé à l'émétique ou à l'atoxyl. *Ibid.*, XXIV, 220, 234.

THOMAS, H. W. (1905). Some experiments in the treatment of trypanosomiasis. *Brit. Med. Journ.*, I, 1140.

THOMSON, D. (1914). A demonstration on the cultivation of malarial parasites (*Plasmodium falciparum* and *Plasmodium vivax*). *Trans. Soc. Trop. Med. & Hyg.*, VII, 126.

THOMSON, D. B., & BALFOUR, A. (1910). Two cases of non-ulcerating "oriental sore," better termed Leishman nodules. *Ibid.*, III. 107. *Journ. R. Army Med. Corps*, XIV, 1.

THOMSON, J. D. (1906). Blood parasites of the mole, including a new form of intracorpuscular parasite. *Journ. Hyg.*, VI, 574.

—— (1908). Cultivation of the trypanosome found in the blood of the goldfish. *Ibid.*, VIII, 75.

—— (1917). Notes on malaria. *Journ. R. Army Med. Corps*, XXIX, 379.

—— (1918). Quinine in malaria: its limitations and possibilities. *Trans. Soc. Trop. Med. & Hyg.*, XI, 226.

THOMSON, J. D., & WOODCOCK, H. M. (1922). The parasites of malaria. Byam & Archibald's *Practice of Medicine in the Tropics*, II, 1516. London.

THOMSON [J. G.] (1918). Amœbic dysentery. *Lancet*, II, 451.

—— (1925). A *Giardia* parasitic in a bursate nematode living in the Viscacha. *Protozoology*, No. 1, 1.

THOMSON, J. G. & D. (1913). The cultivation of one generation of benign tertian malarial parasites (*Plasmodium vivax*) in vitro by Bass's method. *Ann. Trop. Med. & Parasit.*, VII, 153.

—— (1913*a*). The growth and sporulation of the benign and malignant tertian malarial parasites in the culture tube and in the human host. *Ann. Trop. Med. & Parasit.*, VII, 509; *Proc. Roy. Soc.*, B, LXXXVII, 77.

THOMSON, J. G., & FANTHAM, H. B. (1913). The culture of *Babesia* (*Piroplasma*) *canis* in vitro. *Ann. Trop. Med. & Parasit.*. VII. 621.

—— —— (1914). The successful cultivation of *Babesia* (*Piroplasma*) *canis* in vitro, following the method of Bass. *Trans. Soc. Trop. Med. & Hyg.*, VII, 119.

THOMSON, J. G.. & LUCAS, C. L. T. (1926). A preliminary note on the study of *Endamœba blattæ* (Butschli, 1878) Leidy, 1879, found parasitic in the intestine of *Blatta orientalis* in England, with some remarks on its generic status. *Journ. Trop. Med. & Hyg.*, XXIX, 41.

THOMSON, J. G., & McLELLAN, S. W. (1912). The cultivation of one generation of malarial parasites (*Plasmodium falciparum*) in vitro by Bass's method. *Ann. Trop. Med. & Parasit.*, VI, 449.

THOMSON, J. G., & ROBERTSON. A. (1921). The value of laboratory reports on stools in cases of suspected amœbic dysentery and their interpretation by the clinician, with a special note on the diagnostic significance of Charcot-Leyden crystals. *Proc. Roy. Soc. Med.*, XIV, Sect. Trop. Dis., 33.

—— —— (1921*a*). Charcot-Leyden crystals in the stools as an aid to the diagnosis of entamœbic dysentery. *Journ. Trop. Med. & Hyg.*, XXIV, 289.

—— —— (1922). A case of human infection with *Eimeria oxyspora* Dobell, 1919. *Ibid.*, XXV, 369.

—— —— (1923). *Dientamœba fragilis*, Jepps and Dobell, 1917. A case of human infection in England. *Ibid.*, XXVI, 135.

—— —— (1925). Notes on the cultivation of certain amœbæ and flagellates of man, using the technique of Boeck and Drbohlav. *Ibid.*, XXVIII, 345.

—— —— (1926). Fish as the source of certain coccidia recently described as intestinal parasites of man. *Brit. Med. Journ.*, 1. 282.

TIBALDI, E. (1920). Sopra una nuova specie di ameba parassita trovata nelle tonsille (*Entamœba macrohyalina*). *Annali d'Igiene*, XXX, 613.

TOBEY. E. N. (1906). Trypanosomiasis of the newt. *Journ. Med. Res.*, XV, 147.

TODD, J. L. (1914). The trypanosome of Gambian mice. *Ann. Trop. Med. & Parasit.*, VIII, 469.

TODD, J. L., & WOLBACH, S. B. (1912). Parasitic Protozoa from the Gambia. *Journ. Med. Res.*, XXVI, 195.

TOENNIGES (1898). Die feineren Bauverhältnisse von *Opalina ranarum*. *Sitzungsb. Ges. z. Beförd. d. Ges. Naturwiss. Marburg*, 126.

TÖPFER, H. (1916). [Discussion following Jungmann's " Zur Aetiologie des ' Febris Wolhynica.' "] *Berlin. Klin. Woch.*, LIII, 323.

—— (1917). Ursache und Übertragung der Kriegsnephritis. *Med. Klin.*, XIII. 678.

TOIT, P. J. DU (1918). Zur Systematik der Piroplasmen. *Arch. Protist.*, XXXIX, 84.

—— (1919). Experimentelle Studien über die Pferdepiroplasmose. I. Mitteilung. Kreuzimpfungsversuche mit *Nuttallia equi* (Laveran. 1901) und *Piroplasma caballi* Nuttall, 1910. *Arch. Schiffs.- u. Trop.-Hyg.*, XXIII, 121.

—— (1919*a*). Experimentelle Studien über die Pferdepiroplasmose. II. Mitteilung. Übertragungsversuche mit *Ixodes ricinus* L. bei der *Nuttallia-equi* Infektion. *Ibid.*, XXIII, 141.

TORRES, M. (1915). Alguns fatos que interessam a epidemiolojia da molestia de Chagas. *Mem. Inst. O. Cruz.*, VII, 120.

TORRES. O. (1920). A case of multiple lesions in leishmaniasis. *New Orleans Med. & Surg. Journ.*, LXXIII, 61.

TOURNIER, E. (1920). Note sur un cas de kala-azar infantile observé au Gabon. *Bull. Soc. Path. Exot.*, XIII, 175.

TOWNSEND, C. H. T. (1915). The insect vector of uta, a Peruvian disease. *Journ. Parasit.*, II, 67.

TRAUTMANN, O. (1922). Über das kulturelle und serologische Verhalten des *Trypanosoma theileri*. (Dissert. Inst Schiffs.- u. Tropenkr. Hamburg.)

TREILLARD, M.. & LWOFF, A. (1924). Sur un infusoire parasite de la cavité générale des larves de chironomes. Sa sexualité. *C. R. Acad. Sci.*, CLXXVIII, 1761.

TRIFFITT, M. J. (1924). Note on an *Eimeria* n. sp. found in the fæces of an eland. *Journ. Trop. Med. & Hyg.*, XXVII, 223.

—— (1925). Observations on *Gastrocystis gilruthi*, a parasite of sheep in Britain. *Protozoology*, No. 1, 7.

—— (1925*a*). Observations on two new species of coccidia parasitic in snakes. *Ibid.*, No. 1, 19.

—— (1926). Some sporozoan parasites found in the intestinal wall of Bennett's wallaby (*Macropus bennetti*). *Ibid.*, No. II, 31.

TWORT, C. C. (1922). Encephalo-myelitis of rabbits. *Vet. Journ.*, LXXVIII, 194.

Tyzzer, E. E. (1907).   A sporozoan found in the peptic glands of the common mouse.   *Proc. Soc. Exp. Biol. & Med.*, V, 12.

—— (1910).   An extracellular coccidium, *Cryptosporidium muris* (gen. et sp. nov.), of the gastric glands of the common mouse.   *Journ. Med. Res.*, XXIII, 487.

—— (1912).   *Cryptosporidium parvum* (sp. nov.), a coccidium found in the small intestine of the common mouse.   *Arch. Protist.*, XXVI, 394.

—— (1919).   Developmental phases of the Protozoon of " blackhead " in turkeys.   *Journ. Med. Res.*, XL, 1.

—— (1920).   Amœbæ of the cæca of the common fowl and of the turkey, *Entamœba gallinarum* sp. n. and *Pygolimax gregariniformis* gen. et spec. nov.   *Ibid.*, XLI, 199.

—— (1920a).   Observations on the transmission of " blackhead " in turkeys.   The common fowl as a source of infection.   *Ibid.*, XLI, 219.

—— (1924).   The chicken as a carrier of *Histomonas meleagridis* (blackhead): the Protozoön in its flagellated stage.   *Ibid.*, XLIV, 676.

Tyzzer, E. E., & Fabyan, M. (1920).   Further studies on " blackhead " in turkeys, with special reference to transmission by inoculation.   *Journ. Infect. Dis.*, XXVII, 207.

Tyzzer, E. E., Fabyan, M., & Foot, N. C. (1921).   Further observations on " blackhead " in turkeys.   *Ibid.*, XXIX, 268.

Tyzzer, E. E., & Walker, E. L. (1919).   A comparative study of *Leishmania infantum* of infantile kala-azar and *Leptomonas* (*Herpetomonas*) *ctenocephali* parasitic in the gut of the dog flea.   *Journ. Med. Res.*, XL, 129.

Vaglio, R. (1914).   Contributo alla ricerca del parassita di Leishman nel sangue periferico di bambini affetti da leishmaniosi.   *Pediatria*, XXII, 682.

Vahlkampf, E. (1905).   Beiträge zur Biologie und Entwicklungsgeschichte von *Amœba limax* einschliesslich der Züchtung auf künstlichen Nährböden.   *Arch. Protist.*, V, 167.

Valentin (1841).   Ueber ein Entozoon im Blute von *Salmo fario*.   *Arch. Anat. Phys. Wiss. Med.*, 435.

Valladares, J. F. (1914).   Equine biliary fever in Madras.   *Parasitology*, VII, 88.

Vandenbranden, F., & Hoof, L. van (1923).   Résultats de l'observation de malades trypanosomés traités au tryparsamide " A 63."   *Bull. Soc. Path. Exot.*, XVI, 606.

Vassal, J. J. (1905).   Sur un hémacytozoaire endoglobulaire nouveau d'un mammifère.   *Ann. Inst. Pasteur*, XIX, 224.

—— (1905a).   Sur un hématozoaire endoglobulaire pigmenté d'un écureuil de l'Annam.   *C. R. Soc. Biol.*, LVIII, 350.

—— (1907).   Sur un hématozoaire d'un chéiroptère.   *Ann. Inst. Pasteur*. XXI, 224.

—— (1907a).   Nouvelle contribution à l'étude de l'hématozoaire de l'écureuil (*Hœmamœba vassali* Lav.).   *Ibid.*, XXI, 851.

Vedder, E. B. (1912).   An experimental study of the action of ipecacuanha on amœbæ.   *Far East. Assoc. Trop. Med. Trans. 2nd Congr.* (Hongkong), 87; *Journ. Trop. Med. & Hyg.*, XV, 313.

Veglia, F. (1915).   Coltura dell'*Anaplasma marginale in vitro*.   (Nota prelim.)   *Giorn. R. Accad. Med. Torino*, LXXVIII, 33.

Velez (1913).   Uta et espundia.   *Bull. Soc. Path. Exot.*, VI, 545.

Velu, H. (1918).   Les affections du cheval à parasites endoglobulaires au Maroc.   *Ibid.*, XI, 26.

—— (1919).   La coccidiose de la chèvre au Maroc et le parasitisme latent de *Eimeria arloingi* *Ibid.*, XII, 298.

—— (1920).   Infection spontanée du chien à *Trypan. marocanum*.   *Ibid.*, XIII, 234.

—— (1921).   La piroplasmose bovine au Maroc et ses rapports avec les piroplasmoses circumméditerranéennes.   *Ibid.*, XIV, 116.

—— (1922).   Rapport sur le fonctionnement du laboratoire de recherches du service de l'élevage du Maroc pendant l'année 1921.   *Arch. Inst. Pasteur Afrique du Nord*, II. 444.

—— (1922a).   Les piroplasmes et les piroplasmoses.   *Mém. Soc. Sci. Nat. Maroc.*, II.

—— (1923).   Contribution à l'étude des maladies à parasites endoglobulaires du bétail marocain.   *Ann. Parasit. Hum. & Comp.*, I, 54.

Velu, H., & Eyraud, R. (1916).   Trypanosomiase des chevaux du Maroc.   Infestation d'un jeune chien par l'allaitement.   *Bull. Soc. Path. Exot.*, IX, 567.

Vialatte, C. (1915).   Au sujet d'un trypanosome du chien observé dans le Sahara Oranais.   *Ibid.*, VIII, 70.

—— (1922).   Essai de coordination de quelques données relatives à la biologie des parasites du paludisme.   *Arch. Inst. Pasteur Afrique du Nord*, II, 341.

Vianna, G. (1911).   Beitrag zum Studium der pathologischen Anatomie der Krankheit von Carlos Chagas (Schizotrypanose des Menschen oder parasitaere Thyreoiditis).   *Mem. Inst. O. Cruz*, III, 276.

—— (1911a).   Algumas notas sobre o cyclo evolutivo do " *Trypanosoma gambiense*."   (Nota preliminar.)   *Brazil-Medico*, XXV, 61.

VIANNA, G. (1911b). Algumas phases de evoloção dos trypanozomas equineo e congolense. *Ibid.*, XXV, 103.

VIERECK, H. (1907). Studien über die in den Tropen erworbene Dysenterie. *Arch. Schiffs.- u. Trop.-Hyg.*, XI (Beih. I), 1.

VIRCHOW, R. (1849). Zur pathologischen Physiologie des Blutes. *Arch. Path. Anat.*, II, 587.

—— (1858). *Die Cellularpathologie in ihrer Begrundung auf physiologische und pathologische Gewebelehre.* Berlin, 198.

—— (1860). Helminthologische Notizen. 3. Ueber *Trichina spiralis. Arch. Path. Anat.*, XVIII, 330.

—— (1860a). Helminthologische Notizen. 4. Zur Kenntnis der Wurmknoten. *Ibid.*, XVIII, 523.

—— (1865). Zur Trichinen-Lehre. *Ibid.*, XXXII, 332.

VOEGTLIN, C., SMITH, H. W., & OTHERS (1920). Quantitative studies in chemotherapy. I. The trypanocidal action of arsenic and antimony compounds. *U.S. Publ. Health Rep.*, XXXV, 2264.

VOGES, O. (1901). Das Mal de Caderas der Pferde in Südamerika. *Berlin. Tierärztl. Woch.*, XVII, 597.

VOLPINO, G. (1911). Infezione sperimentale da *Leishmania infantum* nella cornea del coniglio. *Pathologica*, III, 45.

VRIJBURG, A. (1913). Einige Untersuchungen über *Babesia bigemina. Zeitschr. Infektionskr. d. Haust.*, XIII, 180.

—— (1918). Babesiose en babesiaparasieten in Nederland. *Tijdschr. Diergeneesk.*, XLV, 535, 564.

VULPIAN. [A.] (1858). Sur les corps oviformes que l'on trouve dans les conduits biliaires, la vésicule biliaire, le mucus intestinal et les parois intestinales des lapins et d'autres animaux (chats). *C. R. Soc. Biol.*, V, 186.

WAGENER, E. H. (1923). A skin reaction to extracts of *Leishmania tropica* and *Leishmania infantum. Univ. California Publ. Zool.*, XX, 477.

WALCH, E., & WALCH-SORGDRAGER, R. (1921). A malarial epidemic caused by *M. sinensis. Meded. Burgerlijk. Geneesk. Dienst. Ned.-Indi ·*, 3.

WALDENBURG, L. (1867). Zur Entwicklungsgeschichte der Psorospermien. *Arch. Path. Anat.*, XL. 435.

WALKER, E. L. (1908). The parasitic amœbæ of the intestinal tract of man and other animals. *Journ. Med. Res.*, XVII, 379.

—— (1909). Sporulation in the parasitic Ciliata. *Arch. Protist.*, XVII, 297.

—— (1910). *Trypanoplasma ranæ* n. sp. and its life-cycle in cultures. *Journ. Med. Res.*, XXIII, 391.

—— (1911). A comparative study of the amœbæ in the Manila water supply, in the intestinal tract of healthy persons, and in amœbic dysentery. *Philippine Journ. Sci.*, (B), VI, 259.

—— (1913). Experimental balantidiasis. *Ibid.*, VIII, 333.

WALKER, E. L., & SELLARDS, A. W. (1913). Experimental entamœbic dysentery. *Ibid.*, VIII, 253.

WALRAVENS, P. (1924). La trypanosomiase chez le porc due au *Tryp. rodhaini* (n. sp.). *Bull. Méd. du Katanga*, I, 132.

WALTON, H. J. (1915). A case of urinary amœbiasis. *Brit. Med. Journ.*, I, 844.

WALZBERG, U. (1923). Zur pathologischen Histologie der natürlichen Toxoplasmose des Zeisigs. *Zeitschr. Infektionskr. d. Haust.*, XXV, 19.

WANDOLLECK, B. (1895). *Pyrosoma bigeminum. Centralbl. Bakt.*, I. Abt., XVII, 554.

WARE, F. (1916). The possibility of amœbic dysentery in the dog, and its treatment with emetin. *Journ. Comp. Path. & Therap.*, XXIX, 126.

WARTHIN, A. S. (1922). The occurrence of *Entamœba histolytica* with tissue lesions in the testis and epididymis in chronic dysentery. *Journ. Infect. Dis.*, XXX, 559.

WASIELEWSKI, T. VON (1896). *Sporozoenkunde.* Jena.

—— (1898). Über geisseltragende Coccidienkeime. *Centralbl. Bakt.*, I. Abt., XXIV, 71.

—— (1904). *Studien und Mikrophotogramme zur Kenntnis der pathogenen Protozoen.* I. Untersuchungen über den Bau, die Entwicklung und über der pathogene Bedeutung der Coccidien. Leipzig.

—— (1908). *Studien und Mikrophotogramme zur Kenntnis der pathogenen Protozoen.* II. Untersuchungen über Blutschmarotzer. Leipzig.

WASIELEWSKI, T. VON, & HIRSCHFELD. L. (1910). Untersuchungen über Kulturamöben. *Abh. Heidelberg. Akad. Wiss.* Math.-Naturwiss. Kl., 1.

WASIELEWSKI, T. VON, & WÜLKER. G. (1918). Die Hämoproteus-Infektion des Turmfalken. *Arch. Schiffs.- u. Trop.-Hyg.*, XXII (Beih. 2), 117.

WASSELL, C. M. (1923). *Isospora hominis* discovered in China. *China Med. Journ.*, XXXVII, 661.

WATSON, E. A. (1909). Sarcosporidiosis. *Journ. Comp. Path. & Therap.*, XXII. 1.

—— (1915). Dourine and the complement fixation test. *Parasitology*, VIII, 156.

—— (1920). Dourine in Canada. 1904-1920. History, research, and suppression. *Dept. Agric. Health. Anim. Branch, Dom. Canada*, 1.

WATSON, E. A., & HADWEN, S. (1912). Trypanosomes found in Canadian mammals. *Parasitology*, V, 21.

WATSON. M. (1916). A new infusorian parasite in sand-fleas. *Journ. Parasit.*, II, 145.

WAWORUNTU, F. K. (1924). Bijdrage tot de kennis van het konijnen-coccidium. *Proefschr. Veeartsenijk. Hoogeſch.* Utrecht.

WEBB. E. C. (1906). Piroplasmosis in fox-hounds in India. *Journ. Comp. Path. & Therap.*, XIX, 1.

WECK (1914). Beobachtungen über Trypanosomen des Menschen und der Tiere am Rovuma-Flusse. *Arch. Schiffs.- u. Trop.-Hyg.*, XVIII, 113.

WEIDMAN, F. D. (1915). *Coccidium bigeminum* Stiles in swift foxes (habitat Western U.S.). *Journ. Comp. Path. & Therap.*, XXVIII, 320.

WEIGL. R. (1921). Further studies on *Rickettsia rocha-limœ* (n. s.). *Przeglad Epidem̦.*, Warsaw, I, 375.

WEISSENBERG, R. (1912). *Callimastix cyclopis* n. g., n. sp., ein geisseltragendes Protozoon aus dem Serum von *Cyclops*. *Sitzungsb. Ges. Naturf. Freunde*, 299.

WEISSENBORN, E. (1911). Beitrag zur Kenntnis der kurzgeissligen Trypanosomen. *Arch. Schiffs.- u. Trop.-Hyg.*, XV, 477.

WELCH, W. H. (1897). Malaria: Definition, history, and parasitology. Loomis, *Syst. Pract. Med.*, New York, I, 17.

WELLMAN, C., & WHERRY. W. B. (1910). Some new internal parasites of the California ground squirrel *Otospermophilus beecheyi*. *Parasitology*, III, 417.

WELLMER. L. (1910). Sporozoen ostpreussischer Arthropoden. *Schr. Physik. Ges. Königsberg*, LII. 103.

WELLS. R. T. (1911). Aerial contamination as a fallacy in the study of amœbic infections by cultural methods. *Parasitology*. IV, 204.

WELSH, D. A., & BARLING, J. E. ꞌ. (1909). *Hæmogregarina petauri :* a hæmogregarine of a marsupial flying squirrel. *Trans. Australian Med. Congr.* (1908), II, 329; *Journ. Path. & Bact.*, 1910, XIV, 536.

WELSH. D. A., & DALYELL, E. J. (1910). *Hæmogregarina peramelis :* a free hæmogregarine of an Australian bandicoot. *Journ. Path. & Bact.*, XIV, 547.

WEISH, D. A., DALYELL, E. J., & BURFITT, M. B. (1909). *Hæmogregarina dasyuri :* a hæmogregarine of the Australian native cat. *Trans. Australian Med. Congr.* (1908), II, 333; *Journ. Path. & Bact.*, 1910, XIV, 542.

WENDELSTADT, H., & FELLMER, T. (1909). Einwirkung von Kaltblüterpassagen auf Nagana und Lewisi-Trypanosomen. *Zeitschr. Imm.-Forsch.*, III, 422.

WENRICH. D. H. (1921). The structure and division of *Trichomonas muris* (Hartmann). *Journ. Morph.*, XXXVI, 119.

—— (1923). Variations in *Euglenamorpha hegneri*, n. g., n. sp., from the intestine of tadpoles. *Anatom. Rec.*, XXIV, 370.

WENYON, C. M. (1906). Report on Dr. P. N. Gerrard's preparations accompanying the foregoing communication. *Journ. Hyg.*, VI, 231.

—— (1907). Observations on the Protozoa in the intestine of mice. *Arch. Protist.*, Suppl. I (Festschr. f. R. Hertwig), 169.

—— (1908). A trypanosome and hæmogregarine of a tropical American snake. *Parasitology*, I, 315.

—— (1909). Report of travelling pathologist and protozoologist. *3rd Rept., Wellcome Trop. Res. Labs.. Khartoum*, 121. (Vol. dated 1908, but published 1909.)

—— (1910). Some observations on a flagellate of the genus *Cercomonas*. *Quart. Journ. Micro. Sci.*, LV, 241.

—— (1910a). Some remarks on the genus *Leucocytozoon*. *Parasitology*, III, 63.

—— (1910b). A new flagellate (*Macrostoma mesnili* n. sp.) from the human intestine, with some remarks on the supposed cysts of *Trichomonas*. *Ibid.*, III, 210.

—— (1911). Note on the occurrence of *Herpetomonas* in the *Phlebotomus* of Aleppo. *Journ. London Sch. Trop. Med.*, I, 98.

—— (1911a). Oriental sore in Bagdad, together with observations on a gregarine in *Stegomyia fasciata*, the hæmogregarine of dogs, and the flagellates of house flies. *Parasitology*, IV, 273.

—— (1911-1912). *Kala-azar Bulletin*. Tropical Diseases Bureau, London.

—— (1912). The insufficiency of the posterior nucleus as a specific distinction in *Trypanosoma rhodesiense*. *Journ. Trop. Med. & Hyg.*, XV, 193.

—— (1912a). Some remarks on the successful inoculation of *Leishmania tropica* to man. *Journ. London Sch. Trop. Med.*, I, 224.

WENYON, C. M. (1912*b*). A case of dermal leishmaniasis from South America, with some remarks on the structure of the parasite and its culture. *Ibid.*, I, 207.

—— (1912*c*). Experiments on the behaviour of Leishmania and allied flagellates in bugs and fleas, with some remarks on previous work. *Ibid.*, II, 13.

—— (1912*d*). Experimental amœbic dysentery and liver abscess in cats. *Ibid.*, II, 27.

—— (1913). A further note on a case of dermal leishmaniasis from South America, with the results of inoculation experiments. *Ibid.*, II, 117.

—— (1913*a*). Observations on *Herpetomonas muscœ domesticœ* and some allied flagellates. *Arch. Protist.*, XXXI, 1.

—— (1913*b*). Experiments on the transmission of *Trypanosoma lewisi* by means of fleas. *Journ. London Sch. Trop. Med.*, II, 119.

—— (1914). The culture of Leishmania from the finger blood of a case of Indian kala-azar, with some remarks on the nature of certain granular bodies recently described from this disease. *Journ. Trop. Med. & Hyg.*, XVII, 49.

—— (1914*a*). Kala-azar in Malta, with some remarks on the various leishmaniases. *Trans. Soc. Trop. Med. & Hyg.*, VII, 97.

—— (1915). The pigmented parasites of cold-blooded animals, with some notes on a plasmodium of the Trinidad iguana. *Journ. Trop. Med. & Hyg.*, XVIII, 133.

—— (1915*a*). Flagellate forms of *Leishmania donovani* in the tissues of an experimentally infected dog. *Ibid.*, XVIII, 218.

—— (1915*b*). Leishmania problems. observations on a recent contribution to the subject. *Ibid.*, XVIII, 241.

—— (1915*c*). Report of the protozoologist for the half-year ending 30th April, 1914. *Rept. Advis. Cttee. Trop. Dis. Res. Fund* (1914), 92.

—— (1915*d*). Piroplasmosis of Rhodesian sheep as observed by Bevan. *Journ. Comp. Path. & Therap.*, XXVIII, 60.

—— (1915*e*). Observations on the common intestinal Protozoa of man· their diagnosis and pathogenicity. *Journ. R. Army Med. Corps*, XXV, 600.

—— (1915*f*). The development of the oöcyst of the human coccidium: an addendum. *Lancet*, II, 1296.

—— (1915*g*). Another human coccidium from the Mediterranean war area. *Ibid.*, II, 1404.

—— (1916). The protozoological findings in five hundred and fifty-six cases of intestinal disorder from the Eastern Mediterranean war area. *Journ. R. Army Med. Corps*, XXVI, 445.

—— (1920). Histological observations on the possible pathogenicity of *Trichomonas intestinalis* and *Chilomastix mesnili*, with a note on *Endolimax nana*. *Journ. Trop. Med. & Hyg.*, XXIII, 125.

—— (1921). Observations on the intestinal Protozoa of three Egyptian lizards, with a note on a cell-invading fungus. *Parasitology*, XII, 350.

—— (1921*a*). Laboratory meeting. *Trans. Roy. Soc. Trop. Med. & Hyg.*, XV, 153.

—— (1921*b*). The action of " Bayer 205 " on *Trypanosoma equiperdum* in experimentally infected mice. *Brit. Med. Journ.*, II, 746.

—— (1921*c*). The incidence and aetiology of malaria in Macedonia. *Journ. R. Army Med. Corps*, XXXVII, 81.

—— (1922). Leishmaniasis: a review of recent literature. *Trop. Dis. Bull.*, XIX, 1, 182.

—— (1922*a*). On three supposed new Entamœbæ of man: a review. *Ibid.*, XIX, 19.

—— (1923). " Hæmogregarines " in man, with notes on some other supposed parasites. *Ibid.*, XX, 527.

—— (1923*a*). Coccidiosis of cats and dogs, and the status of the *Isospora* of man. *Ann. Trop. Med. & Parasit.*, XVII, 231.

—— (1925). The genera *Councilmania, Karyamœba*, and *Caudamœba*. *Trop. Dis. Bull.*, XXII, 333.

—— (1926). Coccidia of the genus *Isospora* in cats, dogs and man. *Parasitology*. (In the press).

WENYON, C. M., & BROUGHTON-ALCOCK, W. (1924). A *Trepomonas* coprozoic in human fæces. *Trans. Roy. Soc. Trop. Med. & Hyg.*, XVIII, 9.

WENYON, C. M., & HANSCHELL, H. M. (1913). Notes on *Trypanosoma rhodesiense* from three cases of human trypanosomiasis. *Journ. London Sch. Trop. Med.*, II, 34.

—— (1913*a*). A further note on *Trypanosoma rhodesiense* from three cases of human trypanosomiasis. *Ibid.*, II, 123.

WENYON, C. M., & LOW. G. C. (1914). The occurrence of certain structures in the erythrocytes of guinea-pigs and their relationship to the so-called parasite of yellow fever. *Journ. Trop. Med. & Hyg.*, XVII, 369.

WENYON, C. M., & O'CONNOR, F. W. (1917). *Human intestinal Protozoa in the Near East.* London.

WENYON, C. M., & SCOTT, H. H. (1925). [Demonstration of sections of intestine of Bennett's wallaby.] *Trans. Roy. Soc. Trop. Med. & Hyg.*, XIX, 7.

—— (1925*a*). *Endotrypanum schaudinni* in the two-toed sloth. *Ibid.*, XIX, 280.

WENYON, C. M., & SHEATHER. L. (1925). [*Isospora* infections of dogs.] *Ibid.*, XIX, 10.

WERBITZKI, F. (1910). Über blepharoplastlose Trypanosomen. *Centralbl. Bakt.*, I. Abt., LIII, 303.

WERNER, H. (1908). Studien über pathogene Amöben. *Arch. Schiffs - u. Trop.-Hyg.*, XII (Beih. xi), 419.

—— (1912). *Entamœba coli.* Prowazek's *Handb. d. Path. Prot.*, I, 67.

WESTPHAL, K., & GEORGI (1923). Ueber die Beziehungen der *Lamblia intestinalis* zu Erkrankungen der Gallenwege und Leber. *Münch. Med. Woch.*, LXX. 1080.

WEYGANDT & MÜHLENS (1920). Zur Behandlung der Paralyse. *Ibid.*, LXVII, 884.

WHERRY, W. B. (1913). Studies on the biology of an amœba of the limax group, *Vahlkampfia* sp. No. 1. *Arch. Protist.*, XXXI, 77.

WHINERY, J. B. (1893). Some additional notes on a Myxosporidian infection in the common toad. *New York Med. Journ.*, LVIII, 660.

WHITMORE. E. R. (1911). *Prowazekia asiatica.* (Syn. *Bodo asiaticus* Castellani and Chalmers). *Arch. Protist.*, XXII. 370.

—— (1911a). Parasitäre und freilebende Amöben aus Manila und Saigon und ihre Beziehungen zur Dysenterie. *Ibid.*, XXIII. 71.

—— (1911b). Studien über Kulturamöben aus Manila. *Ibid.*, XXIII, 81. ·

WHITMORE, E. W. (1918). Observations on bird malaria and the pathogenesis of relapse in human malaria. *Johns Hopkins Hosp. Bull.*, No. 325, 62.

WICKWARE, A. B. (1915). Is *Leucocytozoon anatis* the cause of a new disease in ducks ? *Parasitology*, VIII, 17.

WILLACH, P. (1893). Eine durch Infusorien verursachte Taubenepizootie. *Arch. Tierheilk.*, XIX, 36.

WILLIAMSON, R. (1917). A bacteriological examination of convalescents from the Mediterranean Expeditionary Force. Protozoa. *Journ. R. Army Med. Corps*, XXVIII. 451.

WILSON, C. W. (1916). On the life-history of a soil amœba. *Univ. California Publ. Zool.*, XVI, 241.

WILSON, L. B., & CHOWNING. W. M. (1904). Studies in *Piroplasmosis hominis.* ("Spotted fever" or "tick fever" of the Rocky Mountains.) *Journ. Infect. Dis.*, I, 31.

WISING. P. J. (1871). Till kännedomen om *Balantidium coli* hos människan. *Nordisk. Med. Ark.*, III, 1.

—— (1885). [Discussion following communication by Edgren· "Fall af balantidium coli."] *Hygiea*, XLVII (Förhandl. Svensk. Läk.-Sällsk., 48), Stockholm.

WITTICH, VON (1881). Spirillen im Blute von Hamstern. *Centralbl. Med. Wiss.*, XIX. 65.

WLADIMIROFF, A., & YAKIMOFF. W. (1908). Bemerkung zur vorstehenden Mitteilung Wrublewskis. *Centralbl. Bakt.*, I. Abt., XLVIII, 164.

WÖLFEL, K. (1912). Über den derzeitigen Stand der Impfung gegen des Küstenfiebers. *Zeitschr. Infektionskr. d. Haust.*, XII, 247.

WOLBACH, S. B. (1919). Studies on Rocky Mountain spotted fever. *Journ. Med. Res.*, XLI. 1.

WOLBACH. S. B., & BINGER, C. A. L. (1912). A contribution to the parasitology of trypanosomiasis. *Ibid.*, XXVII, 83.

WOLBACH, S. B., CHAPMAN, W. H., & STEVENS, H. W. (1915). Concerning the filterability of trypanosomes. *Ibid.*, XXXIII, 107.

WOLBACH, S. B.. TODD. J. L., & PALFREY, F. W. (1922). *The œtiology and pathology of typhus.* Cambridge, Mass.

WOLTERS, M. (1891). Die Conjugation und Sporenbildung bei Gregarinen. *Arch. Mikr. Anat.*, XXXVII. 99.

WOODCOCK. H. M. (1904). Note on a remarkable parasite of plaice and flounders. *Rept. Lancash. Sea-fisher. Lab.*. 1903, Liverpool, 63.

—— (1904a). Notes on Sporozoa. I. On *Klossiella muris* gen. et spec. nov., Smith and Johnson, 1902. *Quart. Journ. Micro. Sci.*, XLVIII, 153.

—— (1906). The hæmoflagellates· a review of present knowledge relating to the trypanosomes and allied forms. *Ibid.*, L, 151.

—— (1909). The hæmoflagellates and allied forms. Lankester's *Treatise on Zoology.* Part I, London, 193.

—— (1909a). On the occurrence of nuclear dimorphism in a halteridium parasitic in a chaffinch, and the probable connection of this parasite with a trypanosome. *Quart. Journ. Micro. Sci.*, LIII, 339.

—— (1910). Studies on avian Hæmoprotozoa. On certain parasites of the chaffinch (*Fringilla cœlebs*) and the redpoll (*Linota rufescens*). *Ibid.*, LV, 641.

—— (1912). Notes on Sporozoa. Nos. II, III, and IV. *Ibid.*, LVIII, 171.

—— (1914). Further remarks on the flagellate parasites of *Culex.* Is there a generic type, *Crithidia ? Zool. Anz.*, XLIV, 26.

—— (1915). Notes on the Protozoan parasites in the excreta. [Appended to paper by Ledingham & Penfold: Recent bacteriological experiences with typhoidal disease and dysentery.] *Brit. Med. Journ.*, II, 704.

WOODCOCK, H. M. (1916). Observations on coprozoic flagellates, together with a suggestion as to the significance of the kinetonucleus in the binucleata. *Phil. Trans. Roy. Soc.*, B, CCVII, 375.

—— (1921). Notes on coprozoic flagellates. I. On the presence of an accessory flagellum in the genus *Helkesimastix*, Woodcock and Lapage. II. *Alphamonas* Alexeieff, a synonym of *Spiromonas* Perty. *Arch. Zool. Exp.*, LX (N. & R.), 11.

—— (1923). "Rickettsia" bodies as a result of cell digestion or lysis. *Journ. R. Army Med. Corps*, XL, 81, 241.

WOODCOCK, H. M., & LAPAGE, G. (1913). On a remarkable new type of protistan parasite. *Quart. Journ. Micro. Sci.*, LIX, 431.

—— —— (1915). Observations on the life-cycle of a new flagellate, *Helkesimastix fœcicola* n. g., n. sp., together with remarks on the question of syngamy in the trypanosomes. *Proc. Roy. Soc.*, B, LXXXVIII, 353.

WOODCOCK, H. M., & LODGE, O. (1921). Protozoa. Part I. Parasitic Protozoa. *Brit. Antarc.* ("*Terra Nova*") *Expedit.*, *1910, Nat. Hist. Rept. Zool.*, VI, i, 1.

WOODCOCK, H. M., & PENFOLD, W. J. (1916). Further notes on Protozoan infections occurring at the King George Hospital. *Brit. Med. Journ.*, I, 407.

WOODRUFF, L. L. (1917). Rhythms and endomixis in various races of *Paramœcium aurelia*. *Biol. Bull. Marine Biol. Lab. Woods Hole, Mass.*, XXXIII, 51.

—— (1921). The present status of the long-continued pedigree culture of *Paramœcium aurelia* at Yale University. *Proc. Nat. Acad. Sci.*, Washington, VII, 41.

—— (1921a). Micronucleate and amicronucleate races of Infusoria. *Journ. Exp. Zool.*, XXXIV, 329.

—— (1925). The physiological significance of conjugation and endomixis in the Infusoria. *Amer. Natural.*, LIX, 225.

WOODRUFF, L. L., & ERDMANN, R. (1914). A normal periodic reorganization process without cell fusion in *Paramœcium*. *Journ. Exp. Zool.*, XVII, 425.

WOODRUFF, L. L., & MOORE, E. L. (1924). On the longevity of *Spathidium spathula* without endomixis or conjugation. *Proc. Nat. Acad. Sci.*, Washington, X, 183.

WOODRUFF. L. L., & SPENCER, H. (1924). Studies on *Spathidium spathula*. II. The significance of conjugation. *Journ. Exp. Zool.*, XXXIX, 133.

WOODS, A. C., & MORRIS, H. H. (1918). Complement fixation in experimental trypanosomiasis. *Journ. Infect. Dis.*, XXII, 43.

WORDLEY, E. (1921). A new method for the isolation of organisms from fæces and sputum, with some observations on hæmolytic streptococci in fæces obtained by this method. *Journ. Hyg.*, XX, 60.

WRIGHT, E. H. (1920). Preliminary paper on observations on black-water fever (hæmoglobinuria) in the Coorg Province, 1917-18, especially as regards its ætiological factor, namely, a Protozoan parasite of the genus *Piroplasma*, in conjunction with the malarial *Plasmodium*, or a new and undescribed species of *Larerania malariæ* (Donovan's theory). *Ind. Med. Gaz.*, LV, 161.

WRIGHT. J. H. (1903). Protozoa in a case of tropical ulcer ("Delhi sore"). *Journ. Med. Res.*, X, 472.

WRIGHT. J. H., & CRAIGHEAD, E. M. (1922). Infectious motor paralysis in young rabbits. *Journ. Exp. Med.*, XXXVI, 135.

WRUBLEWSKI, K. J. (1908). Ein *Trypanosoma* des Wisent von Bielowesch. *Centralbl. Bakt.*, I. Abt., XLVIII, 162.

YAKIMOFF, W. L. (1910). Die Piroplasmose beim Hunde in Russland. *Zeitschr. Wiss. Prakt. Vet. Med.*, Jurjev, IV, 309.

—— (1910a). Die Zecken und Piroplasmen des Igels. *Centralbl. Bakt.*, I. Abt., LII, 472.

—— (1915). A propos du *Trypanosoma wrublewskyi*. *Bull. Soc. Path. Exot.*, VIII, 431.

—— (1915a). Contribution à l'étude des Leishmanioses de l'homme et du chien dans le Turkestan russe. *Ibid.*, VIII, 474.

—— (1916). A propos de la note de M. Rodhain sur *Theileria ovis*. *Ibid.*, IX, 201.

—— (1917). Parasites du sang des animaux en Transcaucasie. *Ibid.*, X, 98.

—— (1917a). Maladies animales du Turkestan russe à parasites endoglobulaires. A. Piroplasmoses. III. Piroplasmose des moutons. B. Theilérioses. III. Theilériose des chameaux. C. Nuttallioses. II. Nuttalliose des ânes. D. Anaplasmoses. III. Anaplasmose des chiens. *Ibid.*, X, 302.

—— (1921). Sur la question de l'*Uronema caudatum* Martini. *Ibid.*, XIV, 555.

—— (1921a). A propos de l'identification du trypanosome des chameaux du Turkestan russe. *Ibid.*, XIV, 638.

—— (1925). Protistologische Beobachtungen. *Arch. Soc. Russe Protist.*, II, 247.

YAKIMOFF, W. L., & KOHL-YAKIMOFF, N. (1911). Sur un leucocytozoaire du chacal (*Canis adustis*). *Arch. Inst. Pasteur Tunis*, VI, 193.

—— —— (1911a). Observations sur quelques parasites du sang rencontrés au cours de notre mission en Tunisie. *Ibid.*, VI, 198.

YAKIMOFF, W. L., & KOHL-YAKIMOFF, N. (1911b). Un cas de toxoplasme canine en Allemagne. *Bull. Soc. Path. Exot.*, IV, 617

—————— (1911c). La leishmaniose canine. *Arch. Inst. Pasteur Tunis*, VI, 249.

—————— (1912). Infection des souris blanches par les cultures de *Leishmania infantum* Ch. Nicolle. *Bull. Soc. Path. Exot.*, V, 218.

—————— (1912a). L'infection des animaux de laboratoire par la *Leishmania infantum* Ch Nicolle. *Ibid.*, V, 355.

YAKIMOFF, W. L., KOHL-YAKIMOFF, N., & KOBSSAK, D. W. (1910). Hämatoparasitologische Notizen. *Centralbl. Bakt.*, I. Abt., LV, 370.

YAKIMOFF, W. L., & KOLPAKOFF, F. A. (1921). Les colites de l'homme dues aux Protozoaires. *Bull. Soc. Path. Exot.*, XIV, 548.

YAKIMOFF, W. L., & KOSELKINE, P. M. (1917). Maladies animales du Turkestan russe à parasites endoglobulaires. D. Anaplasmoses. II. Anaplasmose des chevaux. *Ibid.*, X, 302.

YAKIMOFF, W. L., & PAROISKY, P. S. (1917). Maladies animales du Turkestan russe à parasites endoglobulaires. B. Theilérioses. II. Theilériose des moutons. *Ibid.*, X, 302.

YAKIMOFF, W. L., & SAPHRONOWITSCH, R. A. (1917). *Theileria* chez le campagnol. *Ibid.*, X, 99.

YAKIMOFF, W. L., & SCHOKHOR, N. I. (1914). Recherches sur les maladies tropicales humaines et animales au Turkestan. I. Répartition de la leishmaniose canine au Turkestan. II. La leishmaniose cutanée (bouton d'Orient) spontanée du chien du Turkestan. *Ibid.*, VII, 185, 186.

—————— (1917). Maladies animales du Turkestan russe à parasites endoglobulaires. A. Piroplasmoses. IV. Piroplasmose de chèvres. V. Piroplasmose des loups. B. Theilérioses. IV. Theilériose des renards. D. Anaplasmoses. I. Anaplasmose des bovidés. *Ibid.*, X, 302.

—————— (1917a). *Leucocytozoon musculi* A. Porter à Pétrograde. *Bull. Soc. Path. Exot.*, X, 100.

YAKIMOFF, W. L., SCHOKHOR, N. I., & KOSELKINE, P. M. (1917). Maladies animales du Turkestan russe à parasites endoglobulaires. A. Piroplasmoses. II. Piroplasmose des chevaux. C. Nuttallioses. I. Nuttalliose des chevaux. *Ibid.*, X, 302.

YAKIMOFF, W. L., & SOLOWZOFF, A. S. (1921). Le *Chilomastix mesnili* Wenyon en Russie. *Ibid.*, XIV, 556.

—————— (1921a). *Monas* sp. dans les selles de l'homme. *Ibid.*, XIV, 557.

YAKIMOFF, W. L., SOLOWZOFF, A. S., & WASSILEWSKY, W. J. (1921). *Oicomonas granulata* n. sp., parasite de l'intestin de l'homme. *Ibid.*, XIV, 546.

YAKIMOFF, W. L., STOLNIKOFF, W. J., & KOHL-YAKIMOFF, N. (1911). Contribution à l'étude de l'*Achromaticus vesperuginis* (Dionisi). *Arch. Protist.*, XXIV, 60.

—————— (1912). Un hémoparasite nouveau des chauves-souris. *Centralbl. Bakt.*, I. Abt., LXII, 283.

—————— (1916). Un parasite nouveau des chauves-souris. *Medicinsk. Obozrenije.*, Moscow, LXXVII, 162.

YAKIMOFF, W. L., WASSILEWSKY, W. J., KORNILOFF, M. T., & ZWIETKOFF, N. A. (1921). Flagellés de l'intestin des animaux de laboratoire. *Bull. Soc. Path. Exot.*, XIV, 558.

YAMASAKI, S. (1924). Über *Leptomonas ctenocephali*, *Trypanosoma lewisi* und pathogene Trypanosomenarten im Hundfloh. *Arch. Protist.*, XLVIII, 136.

YOCOM, H. B. (1918). The neuromotor apparatus of *Euplotes patella*. *Univ. California Publ. Zool.*, XVIII, 337.

YORKE, W. (1911). A note on the pathology of lesions of the cornea and skin in animals experimentally infected with *T. rhodesiense*. *Ann. Trop. Med. & Parasit.*, IV, 385.

—— (1920). Research into the trypanosomiasis problem: a critical consideration of suggested measures. *Trans. Roy. Soc. Trop. Med. & Hyg.*, XIV, 31.

—— (1920a). On human trypanosomiasis in Peru. *Ann. Trop. Med. & Parasit.*, XIII, 459.

YORKE, W., & BLACKLOCK, B. (1911). The trypanosomes found in two horses naturally infected in the Gambia. *Ibid.*, V, 413.

—————— (1914). The differentiation of the more important mammalian trypanosomes. *Ibid.*, VIII, 1.

—————— (1915). The reservoir of the human trypanosome in Sierra Leone. *Ibid.*, IX, 383.

YORKE, W., CARTER, H. F., MACKINNON, D. L., MATTHEWS, J. R., & MALINS SMITH, A. (1917). Persons who have never been out of Great Britain as carriers of *Entamoeba histolytica*. *Ibid.*, XI, 87.

YORKE, W., & MACFIE, J. W. S. (1924). *Trypanosoma evansi* and *Ornithodorus crossi*. *Ibid.*, XVIII, 125.

—————— (1924a). Observations on malaria made during treatment of general paralysis. *Trans. Roy. Soc. Trop. Med. & Hyg.*, XVIII, 13.

YOUNG, C. W., & HERTIG, M. (1926). A search for field and house rodents naturally infected with kala-azar. *Proc. Soc. Exp. Biol. & Med.*, XXIII, 395.

—————— (1925a). Attempts to transmit kala-azar by means of rodent lice, *Haematopinus* sp. *Ibid.*, XXIII, 398.

YOUNG, C. W., & PAO-YUNG LIU (1926). Susceptibility of field, house and laboratory rodents to infection with *Leishmania donovani*. *Ibid.*, XXIII, 392.

YOUNG, C. W., SMYLY, H. J., & BROWN, C. (1924). Experimental kala-azar in a hamster (*Cricetulus griseus*, M.-Edw.). *Ibid.*, XXI, 357.

ZABEL, E. (1901). *Megastoma intestinale* und andere Parasiten in den Zotten eines Magenkrebses. *Arch. Verdauungskr.*, VII, 509.

—— (1904). Flagellaten im Magen. *Wien. Klin. Woch.*, XVII, 1007.

—— (1910). Eiterüberschwemmung des Magendarmkanals aus Nasennebenhöhlenempyemen, nebst einer Bemerkung über die Bedeutung des Flagellatenbefundes im Magen. *Deutsch. Med. Woch.*, XXXVI, 797.

ZACHARIAS, O. (1909). Parasitische Amöben in *Volvox minor*. *Arch. Hydr. u. Plank.*, V, 69.

ZAPFE, H. (1923). Zur Kenntnis der Kokzidiose des Hundes. (Inaug. Diss. Tierärztl. Hochsch. Berlin.)

ZIEMANN, H. (1898). *Über Malaria und andere Blutparasiten*. Jena.

—— (1900). Zweiter Bericht über Malaria und Moskitos an der afrikanischen Westküste. *Deutsch. Med. Woch.*, XXVI, 753. 769.

—— (1900a). Ueber die Beziehungen der Mosquitos zu den Malariaparasiten in Kamerun. *Ibid.*, XXVI, 399.

—— (1902). Über Lomadera, eine Art ausserst verbreiteten Texasfiebers in Venezuela. *Ibid.*, XXVIII, 385.

—— (1902a). Ueber das Vorkommen von *Filaria perstans* und von Trypanosomen beim Chimpanse. *Arch. Schiffs.- u. Trop.-Hyg.*, VI, 362.

—— (1905). Beitrag zur Trypanosomenfrage. *Centralbl. Bakt.*, I. Abt., XXXVIII, 307, 429.

—— (1913). Über die Kultur der Malaria-parasiten und der Piroplasmen (*Piroplasma canis*) in vitro. *Arch. Schiffs.-u. Trop.-Hyg.*, XVII, 361.

—— (1914). Weiteres über die Züchtung der Malariaparasiten und der Piroplasmen (*Piroplasma canis*) in vitro. *Ibid.*, XVIII, 77.

—— (1915). Ueber eigenartige Malariaparasitenformen. *Centralbl. Bakt.*, I. Abt., LXXVI, 385.

—— (1925). Einige Bemerkungen zur Balantidium-coli-Infektion bei Menschen und Schimpansen. *Arch. Schiffs.- u. Trop.-Hyg.*, XXIX, (Beih. 1). 434

ZOTTA, G. (1912). Sur un flagellé du type "*Herpetomonas*" chez *Pyrrhocoris apterus*. *Ann. Scient. Univ. Jassy*, VII, 210.

—— (1921). Sur la culture en milieu N.N.N. du *Leptomonas pyrrhocoris*. *C. R. Soc. Biol.*, LXXXIV, 822.

—— (1921a). Sur la transmission expérimentale du *Leptomonas pyrrhocoris* Z. chez des insectes divers. *Ibid.*, LXXXV, 135.

—— (1923). Emploi de la substance cérébrale comme milieu de culture pour le *Leptomonas pyrrhocoris*. *Ibid.*, LXXXVIII, 281.

—— (1923a). Culture du *Leptomonas pyrrhocoris* en milieu d'organes stérilisés. *Ibid.*, LXXXVIII, 283.

—— (1923b). *Leptomonas familiaris* n. sp., parasite du tube digestif de *Lygaeus familiaris* en Roumanie. *Ibid.*, LXXXVIII, 285.

ZSCHOKKE, E. (1892). Beobachtungen über die rothe Ruhr. *Schweiz. Arch. Tierheilk.*, XXXIV, 49.

—— (1913). Ein Rhinosporidium beim Pferd. *Ibid.*, LV, 641.

ZÜBLIN, E. (1908). Beitrag zur Kenntnis der roten Ruhr des Rindes (Dysenteria coccidiosa bovis). *Ibid.*, L, 123.

ZÜRN, F. A. (1878). *Die kugel- und eiförmigen Psorospermien als Ursache von Krankheiten bei Haustieren*. Leipzig.

ZULUETA, A. (1917). Promitosis y sindiéresis dos modos de división nuclear coexistentes en Amebas del grupo *limax*. *Trabaj. Mus. Cienc. Nat.*, Madrid (Ser. Zool.), XXXIII, 1.

SPIROCHÆTES.

ADDISON, W. (1861). On changes of form in the red corpuscles of human blood. *Quart. Journ. Micro. Sci.*, I, 81.

ARISTOWSKY, W., & HÖLTZER, R. (1924). Bemerkungen zur Morphologie der *Spirochæta obermeieri*. *Centralbl. Bakt.*, I. Abt., XCI, 175.

ARNHEIM, G. (1914). Spirochätenuntersuchungen. *Zeitschr. Hyg. u. Infekt.*, LXXVI, 407.

ARZT, L., & KERL, W. (1914). Ueber experimentelle Kaninchensyphilis und ihre praktische Bedeutung. *Wien. Klin. Woch.*, XXVII, 785.

—— —— (1914a). Weitere Mitteilungen über Spirochätenbefunde bei Kaninchen. *Ibid.*, XXVII, 1053.

BALFOUR, A. (1907). A peculiar blood condition, probably parasitic, in Sudanese fowls. *Journ. Trop. Med. & Hyg.*, X, 153.

—— (1911). The rôle of the infective granule in certain protozoal infections as illustrated by the spirochætosis of Sudanese fowls. *Ibid.*, XIV, 113.

—— (1914). Notes on the life-cycle of the Sudan fowl spirochæte. *Trans. XVII Internat. Congr. Med.*, London, Sect. 21, Part 2, 275.

BALL, V., & ROQUET. M. (1911). Spirochètes et affections hémorrhagiques gastro-intestinales du chien. *Journ. Med. Vét. & Zoot.*, XV, 257.

BATES, L. B., DUNN, L. H., & ST. JOHN, J. H. (1921). Relapsing fever in Panama. The human tick, *Ornithodorus talaje*, demonstrated to be the transmitting agent of relapsing fever in Panama by human experimentation. *Amer. Journ. Trop. Med.*, I, 183.

BATES, L. B., & ST. JOHN, J. H. (1922). Suggestion of *Spirochæta neotropicalis* as name for spirochæte of relapsing fever found in Panama. *Journ. Amer. Med. Assoc.*, LXXIX, 575.

BAYON, H. (1913). A new species of *Treponema* found in the genital sores of rabbits. *Brit. Med. Journ.*, II, 1159.

BIZZOZERO. G. (1893). Sulla presenza di batteri nelle ghiandole rettali, e nelle ghiandole gastriche del cane. *Atti R. Accad. Sci. Torino*, XXVIII, 249.

BLAKE, F. G. (1916). The ætiology of rat-bite fever. *Journ. Exp. Med.*, XXIII, 39.

BLANCHARD. M., & LEFROU, G. (1922). Présence de spirochètes dans le sang d'européens atteints de fièvre bilieuse hémoglobinurique. Le problème étiologique de cette spirochétose. *Bull. Soc. Path. Exot.*, XV, 699.

—— —— (1923). Fièvre bilieuse hémoglobinurique et spirochètes. [Announcement by Mesnil of spirochætes in a third case of blackwater fever.] *Ibid.*, XVI, 297.

BLANCHARD, M., LEFROU, G., & LAIGRET, J. (1922). Note préliminaire sur des cas d'ictères épidémiques observés à Brazzaville (A.E.F.). *Ibid.*, XV, 385.

—— —— —— (1923). Spirochétose ictérigène épidémique observée sur des indigènes à Brazzaville (Afrique Equatoriale Française). *Ibid.*, XVI, 184.

BLANCHARD, R. (1906). Spirilles, spirochètes et autres microorganismes à corps spiralé. *Arch. Parasit.*, X, 129; *Semaine Méd.*, XXVI, 1.

BLIECK. L. DE (1913). Een geval van Spirochætosis bij het rund. *Veeartsenijk. Meded. Dept. Landbouw. Nijv. en Handel.*, No. 8.

BONNE, C. (1924). Conservation du spirochète ictéro-hémorragique dans les punaises. *C. R. Soc. Biol.*, XCI, 242.

—— (1924a). Recherches sur les spirochètes de la spirochétose fébrile de Sumatra. *Bull. Soc. Path. Exot.*, XVII, 623.

BORREL, A. (1905). Infection vermineuse et spirochètes chez les souris cancéreuses. *C. R. Soc. Biol.*, LVIII, 770.

BOSSELUT, R. (1925). Sur un spirochète sanguicole du chien domestique. *Bull. Soc. Path. Exot.*, XVIII, 702.

BREINL, A. (1907). On the morphology and life-history of *Spirochæta duttoni*. *Ann. Trop. Med. & Parasit.*, I, 435.

BREINL, A., & KINGHORN, A. (1906). A preliminary note on a new spirochæta found in a mouse. *Lancet*, II, 651.

BRUCE, D., HAMERTON, A. E., BATEMAN, H. R., MACKIE, F. P., & LADY BRUCE (1911). The occurrence of a spirochæte in the blood of an African antelope. *Rep. Sleep. Sickn. Comm. Roy. Soc.*, XI, 184.

BRUMPT, E. (1908). Existence de la " fièvre des tiques " en Abyssinie. Quelques mots sur la biologie de l'*Ornithodorus moubata*, acarien qui détermine cette spirochétose. *Bull. Soc. Path. Exot.*, I, 432.

—— (1908a). Transmission du *Spirochæta duttoni* par l'*Ornithodorus savignyi*. Transmission du *Spirochæta duttoni* et du *Spirochæta gallinarum* par l'*Ornithodorus moubata*, non transmission des spirochètes de la fièvre récurrente américaine et algérienne par ce même parasite. *Ibid.*, I, 577.

—— (1909). Sur une nouvelle spirochétose des poules du Sénégale produite par *Spirochæta neveuxi* n. sp., *Ibid.*, II, 285.

—— (1909a). Existence d'une spirochétose des poules à *Spirochæta gallinarum* dans le pays Somali. *C. R. Soc. Biol.*, LXVII, 174.

—— (1913). *Précis de parasitologie*. 2nd edition. Paris.

—— (1919). Existence de la spirochétose des bovidés au Brésil. Transmission de cette affection par la tique *Margaropus australis* (Fuller). *Bull. Soc. Path. Exot.*, XII, 748.

—— (1922). *Précis de parasitologie*. 3rd edition. Paris.

BRUMPT, E., & FOLEY (1908). Existence d'une spirochétose des poules à *Spirochæta gallinarum* R. Bl. dans le Sud-Oranais. Transmission de cette maladie par *Argas persicus*. *C. R. Soc. Biol.*, LXV, 132.

BUCHANAN, G. (1924). Spirochætosis ictero-hæmorrhagica. *Brit. Med. Journ.*, II, 990.

BUEN, S. DE. (1926). Note préliminaire d'une fièvre récurrente à tiques, en Espagne. *Bull. Acad. Med.*, XCV, 294.

CARBO-NOBOA, J. M. (1924). Etiologia del dengue. *Ann. Soc. Med.-Quir. Guayas*, Guayaquil. IV, 326.

CARPANO, M. (1912). Spirillosi equina. Un caso di *Spirochæta equi* in un cavallo della colonia Eritrea. *Annali d'Igiene*, XXII, 213.

CARTER, H. V. (1887). Note on occurrence of a minute blood spirillum in an Indian rat. *Sci. Mem. Med. Off. Army India*, Part III, 45.

CARTER, R. M. (1907). The presence of *Spirochæta duttoni* in the ova of *Ornithodorus moubata*. *Ann. Trop. Med. & Parasit.*, I, 155.

CASTELLANI, A. (1905). On the presence of spirochætes in some cases of parangi (yaws, framboesia tropica). Preliminary note. *Journ. Ceylon Branch Brit. Med. Assoc.*, II, 38.

—— (1905a). On the presence of spirochætes in two cases of ulcerated parangi (yaws). *Brit. Med. Journ.*, II, 1280.

—— (1905b). Further observations on parangi (yaws). *Ibid.*, II, 1330.

—— (1906). Note on a peculiar form of hæmoptysis with presence of numerous spirochætes in the expectoration. *Lancet*, I, 1384.

—— (1909). Bronchial spirochætosis. *Brit. Med. Journ.*, II, 782.

CASTELLANI, A., & CHALMERS, A. J. (1910). *Manual of tropical medicine*. 1st edition. London.

CERTES, A. (1882). Notes sur les parasites et les commensaux de l'huitre. *Bull. Soc. Zool.*, Paris, VII, 347.

CHALMERS, A. J., & O'FARRELL, W. R. (1913). Bronchial spirochætosis. *Journ. Trop. Med. & Hyg.*, XVI, 329.

CHATTON, E. (1912). *Treponema drosophilæ* n. sp. Agglutination par le suc des cellules intestinales de l'hôte et cytolyse. *C. R. Soc. Biol.*, LXXIII, 212.

CLELAND, J. B. (1909). On the ætiology of ulcerative granuloma of the pudenda. *Journ. Trop. Med. & Hyg.*, XII, 143.

COHN, F. (1875). Untersuchungen über Bakterien. *Beitr. z. Biol. d. Pflanz.*, I, iii, 141.

COLES, A. C. (1914). Blood parasites found in mammals, birds, and fishes in England. *Parasitology*, VII, 17.

—— (1918). *Spirochæta icterohæmorrhagiæ* in the common rat in England. *Ibid.*, XI, 1.

COOK, A. R. (1904). Relapsing fever in Uganda. *Journ. Trop. Med. & Hyg.*, VII, 24.

COURMONT, J., & DURAND, P. (1917). La spirochétose ictérohémorragique chez le chien. *C. R. Soc. Biol.*, LXXX, 275.

COUVY, L. (1921). Dengue constation de spirochètes dans le sang. *Bull. Soc. Path. Exot.*, XIV, 198.

—— (1921a). Etiologie de la fièvre à phlébotomes. *Ibid.*, XIV, 447.

—— (1922). Note sur deux épidémics de dengue à Beyrouth (1920-21). *Ann. Ist. Pasteur*, XXXVI, 851.

CUNNINGHAM, J. (1925). Serological observations on relapsing fever in Madras. *Trans. Roy. Soc. Trop. Med. & Hyg.*, XIX, 11.

DELAMARE, G., & ACHITOUV (1924). Evolution morphologique de spirochètes fécaux cultivés en bouillon-sérum de cheval. *C. R. Acad. Sci.*, CLXXVII, 1331.

DEUTZ (1912). Über Versuche zur Übertragung von Huhnerspirochaten auf Mause. *Hyg. Rundschau*, XXII, 1017.

DIETERICH, F. H. (1924). The filterability of *Leptospira icteroides* in culture. *Amer. Journ. Trop. Med.*, IV, 553.

DOBELL, C. (1910). On some parasitic Protozoa from Ceylon. *Spolia Zeylanica*, VII, 65.

—— (1911). On *Cristispira veneris* n. sp., and the affinities and classification of spirochætes. *Quart. Journ. Micro. Sci.*, LVI, 507.

—— (1912). Researches on the spirochætes and related organisms. *Arch. Protist.*, XXVI, 117.

—— (1912a). On the systematic position of the spirochætes. *Proc. Roy. Soc.*, B, LXXXV, 186.

DODD, S. (1906). A preliminary note on the identity of the spirochæte found in the horse, ox, and sheep. *Journ. Comp. Path. & Therap.*, XIX, 318.

—— (1910). Spirochætosis in fowls in Queensland. *Ibid.*, XXIII, 1.

DOFLEIN, F. (1911). *Probleme der Protistenkunde*. II. Die Natur der Spirochaten. Jena.

DSCHUNKOWSKY, E. (1912). Das Ruckfallfieber in Persien. *Medicinsk. Obozrenije*, Moscow, LXXVII, 995; *Deutsch. Med. Woch.*, 1913, XXXIX, 419.

DSCHUNKOWSKY, E., & URODSCHEVITCH, J. (1923). The spirochætosis of fowls in Macedonia. *Trans. Roy. Soc. Trop. Med. & Hyg.*, XVI, 478.

DUBOSCQ, O., & LEBAILLY, C. (1912). Sur les spirochètes des poissons. *C. R. Acad. Sci.*, CLIV, 662.

—— (1912a). *Spirella canis* n. g., n. sp., spirille de l'estomac du chien. *Ibid.*, CLIV, 835.

—— (1913). Sur les spirochètes des poissons. (Deuxième note.) *Arch. Zool. Exp.*, LII (N. & R.), 9.

DUJARRIC DE LA RIVIÈRE, M. R. (1918). Contribution à l'étude des spirochétoses—le sodoku. *Ann. Méd.*, V, 184.

DUNKIN, [G. W.] & LAIDLAW [P. P.] (1926).   *Leptospira icterohæmorrhagiæ* in a fox.   *Rept. Med. Research Council, 1924-1925*, 31.

DUNN, L. H. (1923).   The tropical bed bug in relation to the transmission of relapsing fever in Panama.   *Amer. Journ. Trop. Med.*, III, 345.

DUTTON, J. E., & TODD, J. L. (1905).   The nature of human tick fever in the eastern part of the Congo Free State.   *Brit. Med. Journ.*, II, 1259; *Mem. Liverpool Sch. Trop. Med.*, No. XVII.

—— —— (1907).   A note on the morphology of *Spirochæta duttoni. Lancet*, II, 1523.

DUTTON, J. E., TODD, J. L., & TOBEY, E. N. (1906).   Concerning hemoflagellates of an African fish (*Clarias angolensis*).   *Journ. Med. Res.*, XV, 491.

—— —— —— (1906*a*).   Concerning certain parasitic Protozoa observed in Africa.   *Mem. Liverpool Sch. Trop. Med.*, No. XXI, 87.

—— —— —— (1907).   Concerning certain parasitic Protozoa observed in Africa.   *Ann. Trop. Med. & Parasit.*, I, 287.

EDKINS, J. S. (1923).   *Spirilla regaudi* in the cat.   *Parasitology*, XV, 296.

EHRENBERG, H. (1834).   Dritte Beitrag zur Erkenntniss grosser Organisation in der Richtung des kleinsten Raumes.   *Abh. Akad. Wiss. Berlin*, 145.

ETCHEGOIN, E. (1923).   Présence des spirochétidés dans les crachats hémoptoïques.   *C. R. Soc. Biol.*, LXXXIX, 1150.

FANTHAM, H. B. (1908).   *Spirochæta (Trypanosoma) balbianii* (Certes) and *Spirochæta anodontæ* (Keysselitz): their movements, structure, and affinities.   *Quart. Journ. Micro. Sci.*, LII, 1.

—— (1911).   Some researches on the life-cycle of spirochætes.   *Ann. Trop. Med. & Parasit.*, V, 479

—— (1915).   *Spirochæta bronchialis*, Castellani, 1907, together with remarks on the spirochætes of the human mouth.   *Ibid.*, IX, 391.

FARIA, J. G. DE (1924).   *Leptospira couryi*, chez un malade suspect de dengue.   *C. R. Soc. Biol.*, XC, 55.

FRAENKEL, C. (1907).   Beobachtungen an den Spirillen des Zeckenfiebers und des amerikanischen Rekurrens.   *Hyg. Rundschau.* XVII, 263.

FRANCHINI, G. (1924).   Observations sur les hématozoaires des oiseaux d'Italie.   *Ann. Inst. Pasteur*, XXXVIII, 470.

—— (1924*a*).   Hématozoaires particuliers d'un oiseau (*Hypoleis hypoleis*).   *Bull. Soc. Path. Exot.*, XVII, 884.

FÜLLEBORN, [F.], & MAYER, M. (1908).   Über die Möglichkeit der Übertragung pathogener Spirochaeten durch verschiedene Zeckenarten.   *Arch. Schiffs.- u. Trop.-Hyg.*, XII, 31.

FUTAKI, K., TAKAKI, I., TANIGUCHI, T., & OSUMI, S. (1916).   The cause of rat-bite fever.   *Journ. Exp. Med.*, XXIII, 249.

—— —— —— —— (1917).   *Spirochæta morsus muris*, n. sp., the cause of rat-bite fever.   *Ibid.*, XXV, 33.

GALLI-VALERIO, B. (1908).   Spirochétiase des poules déterminée à Lausanne avec *Argas persicus* Fischer de Tunisie.   *Centralbl. Bakt.*, I. Abt., XLVII, 494.

GASPERI, F. DE (1912).   Présence d'un spirochète dans le sang d'un cobaye.   *Bull. Soc. Path. Exot.*, V, 589.

GERLACH, F. (1925).   Geflügelspirochätose in Oesterreich.   *Centralbl. Bakt.*, I. Abt., XCIV, 45.

GILRUTH, J. A. (1910).   Note on the existence of spirochætosis affecting fowls in Victoria.   *Proc. Roy. Soc. Victoria*, XXIII, 102.

GONDER, R. (1907).   Studien über die Spirochäte aus dem Blute von *Vesperugo kuhlii* (Key. u. Blas [Natterer]).   *Arb. K. Gesundheitsamte*, XXVII, 406.

—— (1908).   Spirochäten aus dem Darmtraktus von Pinna.   *Spirochæte pinnæ* nov. spec. und *Spirochæte hartmanni* nov. spec.   *Centralbl. Bakt.*, I. Abt., XLVII, 491.

—— (1914).   Experimentelle Studien über *Spironema gallinarum* und *Spironema recurrentis*.   *Zeitschr. Imm.-Forsch.*, XXI, 309.

GROSS, J. (1910).   *Cristispira* nov. gen.   Ein Beitrag zur Spirochätenfrage.   *Mitt. Zool. Stat. Neapel*, XX, 41.

—— (1912).   Ueber Systematik, Struktur und Fortpflanzung der Spironemacea.   *Centralbl. Bakt.*, I. Abt., LXV, 83.

GROVAS, P. P. (1921).   Experimental transmission of yellow fever.   Cases occurring in Vera Cruz in 1920, together with the isolation of *Leptospira icteroides.   Journ. Amer. Med. Assoc.*, LXXVI, 362.

HAROLD, C. H. H. (1922).   Tick fever in East Persia.   *Journ. R. Army Med. Corps*, XXXVIII, 398.

HATA, S. (1913).   A contribution to our knowledge of the cultivation of *Spirochæta recurrens* [*recurrentis*].   *Centralbl. Bakt.*, I. Abt., LXXII, 107.

HEANLEY, C. M. (1906). A note on the presence of a spirochæta in Chinese buffaloes. *Journ. Comp. Path. & Therap.*, XIX, 322.

HENRY, H. (1910). On the Hæmoprotozoa of British sea fish. (A preliminary note.) *Journ. Path. & Bact.*, XIV, 463.

—— (1912). *Spirochæta pollachii*: a new blood-inhabiting spirochæte from *Gadus pollachius*. the pollack, with a note on the occurrence of certain intracorpuscular bodies in the blood of the Gadidæ. *Ibid.*, XVII, 160.

HINDLE, E. (1911). On the life-cycle of *Spirochæta gallinarum*. Preliminary note. *Parasitology*. IV, 463.

—— (1912). The inheritance of spirochætal infection in *Argas persicus*. *Proc. Cambridge Philos. Soc.*, XVI, 457.

—— (1925). Leptospira in London waters. *Brit. Med. Journ.*, II, 57.

HOGUE, M. J. (1923). *Spirochæta eurygyrata*. A note on its life-history and cultivation. *Journ. Exp. Med.*, XXXVI, 617.

HOLLANDE, A. C. (1921). Présence d'un spirochétoïde nouveau, *Cristispirella caviæ* n. g., n. sp., à membrane ondulante très développée, dans l'intestin du cobaye. *C. R. Acad. Sci.*, CLXXII, 1693.

HUEBENER & REITER (1915). Beiträge zur Etiologie der Weilschen Krankheit. *Deutsch. Med. Woch.*, XLI, 1275.

IDO, Y., ITO, H., & WANI, H. (1918). *Spirochæta hebdomadis*, the causative agent of seven-day fever (Nanukayami). *Journ. Exp. Med.*, XXVIII, 435; XXIX, 199.

INADA, R., & IDO, Y. (1915). A report on the discovery of the causal organism (a new species of spirochæte) of Weil's disease. *Tokyo Ijishinshi*, No. 1908.

ISHIWARA, K., OHTAWARA, T., & TAMURA, K. (1917). Experimental rat-bite fever. *Journ. Exp. Med.*, XXV, 45.

JAFFÉ, J. (1907). *Spirochæta culicis* n. sp. *Arch. Protist.*, IX, 100.

JANCSO, N. (1918). Experimentelle Untersuchungen bezuglich der Pathogenese der Rezidive des Rückfallfiebers. *Centralbl. Bakt.*, I. Abt., LXXXI, 457.

JOEKES, T. (1925). Cultivation of the spirillum of rat-bite fever. *Lancet*, II, 1225.

JOWETT, W. (1910). Note on the occurrence of fowl spirochætosis at the Cape. *Agric. Journ. Cape of Good Hope*, 37.

KASAI, K., & KOBAYASHI, R. (1919). The stomach spirochæte occurring in mammals. *Journ. Parasit.*, VI, 1.

KERMORGANT, Y. (1925). Contribution à l'étude de l'étiologie des oreillons. *Ann. Inst. Pasteur*, XXXIX, 565.

KEYSSELITZ, G., & MAYER, M. (1909). Ueber das Ulcus tropicum. *Arch. Schiffs.- u. Trop.-Hyg.*, XIII, 137.

KING, W. E., BAESLACK, F. W., & HOFFMANN, G. L. (1913). Studies on the virus of hog cholera. *Journ. Infect. Dis.*, XII, 206.

KLARENBEEK, A. (1921). Recherches expérimentales avec un spirochète, se trouvant spontanément chez le lapin et ressemblant au *Treponema pallidum*. *Ann. Inst. Pasteur*, XXXV, 326.

KLEBS, G. (1892). Flagellatenstudien—I & II. *Zeitschr. Wiss. Zool.*, LV, 265, 353.

KLEINE, F. K., & ECKARD, B. (1913). Ueber die Lokalisation der Spirochäten in der Rückfallfieberzecke (*Ornithodorus moubata*). *Zeitschr. Hyg. u. Infekt.*, LXXIV, 389.

KLIGLER, I. J., & ROBERTSON, O. H. (1922). The cultivation and biological characteristics of *Spirochæta obermeieri* (*recurrentis*). *Journ. Exp. Med.*, XXXV, 303.

KNOWLES, R., & DAS GUPTA, B. M. (1924). On a pseudo-organism in the blood in dengue. *Indian Med. Gaz.*, LIX, 11.

KNUTH, P., & TOIT, P. J. DU (1921). Die Spirochätosen. Mense's *Handb. d. Trop. Krankh.*, Leipzig, VI.

KOBAYASHI, R., & KODAMA, M. (1919). A contribution to the study of *Spirochæta morsus muris* in the Nippon field vole (*Microtus montebelli*). *Kitasato Arch.*, III, 199.

KOCH, R. (1905). Vorläufige Mittheilungen über die Ergebnisse einer Forschungsreise nach Ostafrika. *Deutsch. Med. Woch.*, XXXI, 1865.

KŘIVÁČEK, O. (1924). Spirochätenbefunde beim Hundetyphus. *Zeitschr. Hyg. u. Infekt.*, CIII, 529.

KRUMBEIN, R., & FRIELING, B. (1916). Zur Weilschen Krankheit. *Deutsch. Med. Woch.*, XLII, 564.

LAVERAN, A. (1903). Sur la spirillose des bovidés. *C. R. Acad. Sci.*, CXXXVI, 939.

LAVERAN, A., & FRANCHINI, G. (1920). *Herpetomonas* et *Spirochæta* de la blatte orientale. *Bull. Soc. Path. Exot.*, XIII, 331.

—— —— (1921). Spirochétoses de punaises des euphorbes et du latex. *Ibid.*, XIV, 205.

LAVERAN, A., & VALLÉE (1905). Sur un cas de transmission par des ixodes de la spirillose et de la piroplasmose bovines. *C. R. Acad. Sci.*, CXL, 1515.

LAVIER, G. (1921). Les parasites des invertébrés hématophages. *Trav. Lab. Parasit. Fac. Méd. Paris.*

LEBERT, H. (1874). Rückfalltyphus, Flecktyphus und Cholera. Ziemssen's *Handb. Spec. Path. u. Therap.*, II, i, 243.

LEESON, F., & ABBOTT, F. W. (1924). Spirochætes in the blood of wild rats in Accra. *Lab. Journ.*, Liverpool, V, 140.

LEGER, A. (1917). Spirochète de la musaraigne (*Crocidura stampflii* Jentinck). *Bull. Soc. Path Exot.*, X, 280.

—— (1918). Spirochétose sanguine animale à Dakar. Sa valeur au point de vue épidémiologique. *Ibid.*, XI, 64.

LEGER, A., & GALLEN, R. LE (1917). Etude expérimentale du pouvoir pathogène de *Spirochæta crocidurœ*. *Ibid.*, X. 694.

LÉGER, L., & DUBOSCQ, O. (1909). Protistes parasites d'une larve de *Ptychoptera* et leur action sur l'hôte. *Bull. Acad. R. Belgique Sci..* No. 8, 831, 885.

LEGER, M. (1924). Spirochètes sanguicoles, au Sénégal, de l'homme, de la musaraigne et de divers muridés. *Rev. Méd. de Angola*, No. 4, 1923. (No. espec. 1° Congresso de Med. Trop. da Africa ocidental., IV, 279.)

LEISHMAN, W. B. (1907). Observations on the Spirochætæ of relapsing fever and tick fever. *Lancet*, I, 806.

—— (1918). A note on the "granule clumps" found in *Ornithodorus moubata* and their relation to the spirochætes of African relapsing fever (tick fever). *Ann. Inst. Pasteur*, XXXII, 49.

—— (1920). The Horace Dobell Lecture on an experimental investigation of *Spirochæta duttoni*, the parasite of tick fever. *Lancet*, II, 1237.

LEVADITI, C. (1904). Contribution à l'étude de la spirillose des poules. *Ann. Inst. Pasteur*, XVIII, 129.

LEVADITI, C., & LANGE, F. (1905). La spirillose du lapin. Méchanisme de la crise. *C. R. Soc. Biol.*, LVIII, 843.

LEVADITI, C., MARIE, A., & ISAICU, L. (1921). Etude expérimentale de l'hérédité syphilitique. *Ibid.*, LXXXV, 342.

LEVADITI, C., & ROCHÉ, J. (1907). Immunisation des spirilles de la tick fever contre les anti corps. Mécanisme de la rechute. (Deuxième note.) *Ibid.*, LXII, 815.

LINGARD, A. (1907). Some forms of spirochetosis met with in animals in India. *Journ. Trop. Vet. Sci.*, II, 261.

LOW, G. C., & DUNCAN, J. T. (1923). The ætiology of blackwater fever. *Trans. Roy. Soc. Trop. Med. & Hyg.*, XVII, 201.

LÜHE, M. (1906). Spirochäten. Mense's *Handb. d. Trop. Krankh.*, III, 180.

MACFIE, J. W. S. (1914). Notes on some blood parasites collected in Nigeria. *Ann. Trop. Med. & Parasit.*, VIII, 439.

—— (1916). The morphology of certain spirochætes of man and other animals. *Ibid.*, X, 305.

—— (1917). Urethral spirochætosis. *Parasitology*, IX, 274.

MACFIE, J. W. S., & CARTER. H. F. (1917). The occurrence of *Spirochæta eurygyrata* in Europeans in England, with a note on a second species of *Spirochæta* in the human intestine. *Ann. Trop. Med. & Parasit.*, XI, 75.

MACFIE, J. W. S., & YORKE, W. (1917). The relapsing fever spirochætes. *Ibid.*, XI, 81.

MACKIE, F. P. (1907). A preliminary note on Bombay spirillar fever. *Lancet*, II, 832

—— (1907a). The part played by *Pediculus corporis* in the transmission of relapsing fever. *Brit. Med. Journ.*, II, 1706.

MACNEAL, W. J. (1907). A spirochæte found in the blood of a wild rat. *Proc. Soc. Exp. Biol. & Med.*, IV, 125; *Science*, XXVI, 411.

MANTEUFEL (1908). Experimentelle Untersuchungen zur Epidemiologie des europäischen Rück falltiebers. *Arb. K. Gesundheitsamte*, XXIX, 355.

MARCHOUX, E., & COUVY, L. (1913). Argas et spirochètes. Premier mémoire. Les granules de Leishman. Deuxième partie. Le virus chez l'acarien. *Ann. Inst. Pasteur*, XXVII, 450, 620.

MARCHOUX, E., & SALIMBENI, A. (1903). La spirillose des poules. *Ibid.*, XVII, 569.

MARCHOUX, SALIMBENI, & SIMOND (1903). La fièvre jaune. Rapport de la Mission Française. *Ibid.*, XVII, 665.

MARTIN, G. (1906). Sur un cas de spirillose du cheval observé en Guinée française. *C. R. Soc. Biol.*, LX, 124.

MARTOGLIO & CARPANO (1904). Spirillosi ovina. *Annali d'Igiene*, XIV, 577.

MASON, F. E. (1916). Observations on the diseases of Egyptian live stock. *Min. Agric., Vet. Serv., Ann. Rept.*, 1915, Cairo, 34.

MATHIS, C., & LEGER, M. (1911). Spirochète du lapin. *C. B. Soc. Biol.*, LXX, 212.

MAYER, M. (1914). Übertragung von *Spirochæta gallinarum* durch Milben. *Arch. Schiffs.- u. Trop.-Hyg.*, XVIII, 254.

MESNIL, F. (1908). [Review of papers by Brumpt and Doreau.] *Bull. Inst. Pasteur*, VI, 964.

—— (1921). Sur la nature des spirochètes. *La Médecine*, II, 930.

MESNIL, F., & CAULLERY, M. (1916). Sur un organisme spirochétoide (*Cristispira polydoræ* n. sp.) de l'intestin d'une annélide polychète. *C. R. Soc. Biol.*, LXXIX, 1118.

MEZINCESCU, D. (1909). Sur une spirillose du rat. (Note préliminaire.) *Ibid.*, LXVI, 58.

MÖLLERS, B. (1908). Experimentelle Studien über die Übertragung des Rückfallfiebers durch Zecken. *Zeitschr. Hyg. u. Infekt.*, LVIII, 277.

MONTGOMERY, R. E. (1908). On a spirochæte occurring in the blood of chickens in Northern India. *Journ. Trop. Vet. Sci.*, III, 1.

MOOSER, H. (1925). Die Katze als Überträgerin von Sodoku. *Arch. Schiffs.- u. Trop.-Hyg.*, XXIX (Beih. 1), 253.

MÜHLENS, P. (1913). XVI. Rückfallfieber-Spirochäten. XVII. Andere, zum Teil als pathogenen geltende Spirochäten. Kolle & Wassermann's *Handb. d. Path. Mikroorg.*, 2nd edition, VII, 864, 921.

MÜHLENS, P., & HARTMANN, M. (1906). Über *Bacillus fusiformis* und *Spirochæta dentium*. *Zeitschr. Hyg. u. Infekt.*, LV, 81.

NATTAN-LARRIER, L. (1909). Pénétration du spirille de la fièvre récurrente à travers les téguments et les muqueuses intactes. *Bull. Soc. Path. Exot.*, II, 239.

NEIVA, A., CUNHA, A. M. DA, & TRAVASSOS, L. (1914). Contribuições parazitolojicas. *Mem. Inst. O. Cruz*, VI, 180.

NEUMANN, R. O. (1909). Ueber das Verhalten der Spirochäten des Rückfallfiebers in Tierkörper und die experimentelle Uebertragung der Parasiten durch Zecken und Läuse. (Sitz. Naturhist. Med. Ver. Heidelberg, 12 Jan.) *Münch. Med. Woch.*, LVI, 477.

—— (1909a). Studien über protozoische Parasiten im Blute von Meeresfischen. *Zeitschr. Hyg. u. Infekt.*, LXIV, 1.

NEUMANN, R. O., & MAYER, M. (1914). *Atlas und Lehrbuch wichtiger tierischer Parasiten und ihrer Übertrager.* Lehmann's *Med. Atlanten.*, XI, 276.

NICOLLE, C. (1907). Sur une piroplasmose nouvelle d'un rongeur. *C. R. Soc. Biol.*, LXIII, 213.

NICOLLE, C., BLAIZOT, L., & CONSEIL, E. (1912). Conditions de transmisson de la fièvre récurrente par le pou. *C. R. Acad. Sci.*, CLV, 481.

—— —— —— (1913). Etiologie de la fièvre récurrente. Son mode de transmission par les poux. *Ann. Inst. Pasteur*, XXVII, 204.

NICOLLE, C., & COMTE, C. (1905). Sur une nouvelle spirillose. (Note préliminaire.) *C. R. Soc. Biol.*, LIX, 200.

—— —— —— (1906). Sur une spirillose d'une chéiroptère (*l'espertilio kuhlii*). *Ann. Inst. Pasteur*, XX, 311.

NICOLLE, C., & LEBAILLY, C. (1920). Contribution à la connaissance de l'évolution des spirochètes de la fièvre récurrente chez le pou (études des coupes en série). *Arch. Inst. Pasteur Tunis*, XI, 131.

NOC, F. (1920). Les spirochétoses humaines à Dakar (Senegal). *Bull. Soc. Path. Exot.*, XIII, 672.

NOC, F., & STÉVENEL, L. (1913). Flore intestinale du *Stegomyia fasciata* adulte. *Ibid.*, VI, 708.

NOGUCHI, H. (1912). The pure cultivation of *Spirochæta duttoni, Spirochæta kochi, Spirochæta obermeieri*, and *Spirochæta novyi*. *Journ. Exp. Med.*, XVI, 199.

—— (1912a). Cultivation of *Spirochæta gallinarum*. *Ibid.*, XVI, 620.

—— —— (1917). *Spirochæta icterohæmorrhagiæ* in American wild rats and its relation to the Japanese and European strains. *Ibid.*, XXV, 755.

—— (1919). Contribution to the aetiology of yellow fever. *Journ. Amer. Med. Assoc.*, LXXII, 187.

—— (1919a). Transmission experiments on yellow fever. *Journ. Exp. Med.*, XXIX, 565.

—— (1919-1920). Ætiology of yellow fever. *Ibid.*, XXIX, 547; XXX, 1, 13, 87, 401; XXXI, 135; XXXII, 381.

—— (1921). Prophylaxis and serum therapy of yellow fever. *Journ. Amer. Med. Assoc.*, LXXVII, 181.

—— (1921a). A note on the venereal spirochetosis of rabbits. *Ibid.*, LXXVII, 2052.

—— (1922). Researches on yellow fever. Comparative aetiological, pathological, and epidemiological considerations: prevention and treatment. *Lancet*, I, 1185.

—— (1925). Yellow fever research, 1918-1924: a summary. *Journ. Trop. Med. & Hyg.*, XXVIII, 185.

NOGUCHI, H., & KLIGLER, I. J. (1920). Experimental studies on yellow fever occurring in Merida, Yucatan. *Journ. Exp. Med.*, XXXII, 601.

—— —— (1921). Experimental studies on yellow fever in Northern Peru. *Ibid.*, XXXIII, 239.

NOGUCHI, H., MÜLLER, H. R., TORRES, O., SILVA, F., MARTINS, H., DOS SANTOS, A. R., VIANNA, G., & BIÃO, M. (1924). Experimental studies of yellow fever in Northern Brazil. *Monogr. Rockefeller Inst. Med. Res.*, No. 20.

NOVY, F. G. (1907). The rôle of Protozoa in pathology. *Proc. Path. Soc. Philadelphia*, 1.

NOVY, F. G., & KNAPP, R. E. (1906). Studies on *Spirillum obermeieri* and related organisms. *Journ. Infect. Dis.*, III, 291.

NUTTALL, G. H. F. (1908). Note on behaviour of spirochætæ in *Acanthia lectularia*. *Parasitology*, I, 143.

—— (1910). On Hæmatozoa occurring in wild animals in Africa. 2. *Spirochæta bovis caffris* n. sp. found in the buffalo. *Ibid.*, III, 108.

OBERMEIER, O. (1873). Vorkommen feinster, eine eigen Bewegung zeigender Fäden im Blute von Recurrenskranken. *Centralbl. Med. Wiss.*, XI, 145.

OKELL, C. C., DALLING, T., & PUGH, L. P. (1925). Leptospiral jaundice in dogs (yellows). *Vet. Journ.*, LXXXI, 3.

ONORATO, R. (1923). Il sodòku in Tripolitania. *Arch. Ital. Sci. Med. Colon. Tripoli*, IV, 156.

PARKER, H. B., BEYER, G. E., & POTHIER, O. L. (1903). A study of the ætiology of yellow fever. *Yellow Fever Institute Rept. Working Party, No. 1*, Bull. No. 13, 1.

PARMANAND, M. J. (1923). Rat-bite fever, with special reference to its ætiological agent. Preliminary communication. *Indian Journ. Med. Res.*, XI, 181.

PARR, L. W. (1923). Intestinal spirochætes. *Journ. Infect. Dis.*, XXXIII, 369.

PARROT, L. (1920). Spirochètose naturelle du canard domestique. *Bull. Soc. Path. Exot.*, XIII, 647.

PATTON, W. S. (1912). *Spirochæta ctenocephali*, sp. nov., parasitic in the alimentary tract of the Indian dog flea, *Ctenocephalus felis*. *Ann. Trop. Med. & Parasit.*, VI, 357.

PETZOLDT (1915). Kasuistische Mitteilung über einen Fall von Pferde spirillose. *Arch. Schiffs.- u. Trop.-Hyg.*, XIX, 154.

PLAUT, F., & STEINER, G. (1920). Über das Auftreten von Spirosomen und entzündlichen Veränderungen im Liquor bei Rekurrenskranken. *Ibid.*, XXIV, 33.

PLIMMER, H. G. (1912). The President's address On certain blood parasites. *Journ. R. Micro. Soc.*, 133.

PONS, R. (1923). Organisme spiralé mobile isolé en hémoculture au cours d'une fièvre à caractère récurrent. *C. R. Soc. Biol.*, LXXXIX, 1028.

—— (1924). Sur la nature d'un germe spiralé isolé en hémoculture au cours d'une maladie fébrile à caractère récurrent. *C. R. Soc. Biol.*, XC, 396.

PORTER, A. (1910). The structure and life-history of *Crithidia melophagia* (Flu), an endoparasite of the sheep ked, *Melophagus ovinus*. *Quart. Journ. Micro. Sci.*, LV, 189.

PRINGAULT, E. (1921). Présence de spirochètes chez *Phlebotomus perniciosus* Newstead. *C. R. Soc. Biol.*, LXXXIV, 209.

PROWAZEK, S. VON (1907). Vergleichende Spirochaetauntersuchungen. *Arb. K. Gesundheitsamte*, XXVI, 23.

—— (1910). Parasitische Protozoen aus Japan, gesammelt von Herrn Dr. Mine in Fukuoka. *Arch. Schiffs.- u. Trop.-Hyg.*, XIV, 297.

RANKEN, H. S. (1912). Note on the occurrence of a spirochæte in *Cercopithecus ruber*. *Brit. Med. Journ.*, I, 1482.

REANEY, M. F. (1907). Spirillosis of domestic fowls. *Ibid.*, I, 1118.

REED, W., CARROL, J., & AGRAMONTE, A. (1901). The ætiology of yellow fever. An additional note. *Journ. Amer. Med. Assoc.*, XXXVI, 431.

ROAF, H. E. (1922). The Wassermann reaction in relapsing fever. *Brit. Journ. Exp. Path.*, III, 59.

ROBERTSON, A. (1924). Observations on the causal organism of rat-bite fever in man. *Ann. Trop. Med. & Parasit.*, XVIII, 157.

ROBLEDO, E. (1909). Fièvre récurrente de Colombie. *Bull. Soc. Path. Exot.*, II, 117.

ROSS, E. H. (1912). An intracellular parasite developing into spirochætes. *Brit. Med. Journ.*, II, 1651.

ROSS, P. H. (1907). Report on experiments to ascertain the ability of tsetse flies to convey *Trypanosoma gambiense*. *Rep. Sleep. Sickn. Comm. Roy. Soc.*, VIII, 80.

ROSS, P. H., & MILNE, A. D. (1904). Tick fever. *Brit. Med. Journ.*, II, 1453.

ROW, R. (1917). On a new species of spirochæte isolated from a case of rat-bite fever in Bombay. *Indian Journ. Med. Res.*, V, 386.

—— (1918). Cutaneous spirochætosis produced by rat-bite in Bombay. *Bull. Soc. Path. Exot.*, XI, 188.

—— (1922). Some cutaneous manifestations of rat-bite spirochætosis. *Trans. Roy. Soc. Trop. Med. & Hyg.*, XVI, 203.

SAKHAROFF, N. (1891). *Spirochæta anserina* et la septicémie des oies. *Ann. Inst. Pasteur*, V, 564.

SALIMBENI, A. T., KERMORGANT, Y., & GARCIN, R. (1925). La transmission héréditaire du sodoku chez le cobaye. *C. R. Soc. Biol.*, XCIII, 337.

SANGIORGI, G. (1922). Le spirochetosi dei muridi. Nota 2ª (Proposta del sottogenere *Treponemella*). *Pathologica*, XIV, 461.

SCHAUDINN, F. (1904). Generations- und Wirtswechsel bei *Trypanosoma* und *Spirochæte*. *Arb. K. Gesundheitsamte*, XX, 387.

—— (1905). [Correspondence.] *Deutsch. Med. Woch.*, XXXI, 1728.

—— (1907). Zur Kenntnis der *Spirochæta pallida* und anderer Spirochæten. *Arb. K. Gesundheitsamte*, XXVI, 11.

SCHEIN, H. (1910). Spirillose des bovidés dans le Sud-Annam. *Bull. Soc. Path. Exot.*, III, 73.

SCHELLACK, C. (1908). Übertragungsversuche der *Spirochæte gallinarum* durch *Argas reflexus* Fabr. *Centralbl. Bakt.*, I. Abt., XLVI, 486.

—— (1909). Studien zur Morphologie und Systematik der Spirochäten aus Muscheln. *Arb. K. Gesundheitsamte*. XXX, 379.

SCHERESCHEWSKY, J. (1920). Geschlechtlichübertragbare originäre Kaninchensyphilis und Chinin-Spirochätotrophie. *Berlin. Klin. Woch.*, LVII. 1142.

SCHNEIDER (1908). [Remarks after Brumpt's paper.] *Bull. Soc. Path. Exot.*, I. 437.

SCHÜFFNER, W. (1918). Ueber infectiösen Icterus und über einen Spirochätenbefund bei einer klinisch als Schwarzwasserfieber verlaufenen Erkrankung. *Geneesk. Tijdschr. Nederl.-Indië*, LVIII, 352.

SELWYN-CLARKE, P. S., LE FANU, G. H., & INGRAM, A. (1923). Relapsing fever in the Gold Coast. *Ann. Trop. Med. & Parasit.*, XVII, 389.

SERGENT, ED. & ET. (1906). Sur un flagellé nouveau de l'intestin des Culex et des Stegomyia, *Herpetomonas algeriense*. Sur un autre flagellé et sur des *Spirochæte* de l'intestin des larves de moustiques. *C. R. Soc. Biol.*, LX, 291.

SERGENT, ED., & FOLEY, H. (1908). Fièvre récurrente du Sud-Oranais et *Pediculus vestimenti*. Note préliminaire. *Bull. Soc. Path. Exot.*, I, 174.

—— —— (1910). Recherches sur la fièvre récurrente et son mode de transmission, dans une épidémie algérienne. *Ann. Inst. Pasteur*, XXIV, 337.

—— —— (1914). De la période de latence du spirille chez le pou infecté de fièvre récurrente. *C. R. Acad. Sci.*, CLIX, 119.

SIMPSON, J. J. (1914). Entomological research in British West Africa. V. Gold Coast. *Bull. Entom. Res.*, V, 1.

SMITH, G. U. (1909). *On some cases of relapsing fever in Egypt and the question of carriage by domestic vermin*. London.

STEVENSON, A. C. (1922). The incidence of a Leptospira in the kidneys and of parasites in the intestines of one hundred wild rats examined in England. *Amer. Journ. Trop. Med.*, II, 77.

STIMSON, A. H. (1907). Note on an organism found in yellow fever tissues. *U.S. Publ. Health Rep.*, XXII, 541.

ST. JOHN, J. H., & BATES, L. B. (1922). The relapsing fever spirochæte of Panama. Evidence to show that it is a distinct species. *Amer. Journ. Trop. Med.*, II. 251.

STORDY, R. J. (1906). A case of spirillosis in the horse. *Journ. Comp. Path. & Therap.*, XIX, 226.

THEILER, A. (1904). Spirillosis of cattle *Ibid.*, XVII. 47.

—— (1905). Transmission and inoculability of *Spirillum theileri* (Laveran). *Proc. Roy. Soc.*, B, LXXVI, 504.

—— (1909). Transmission des spirilles et des piroplasmes par différentes espèces de tiques. *Bull. Soc. Path. Exot.*, II, 293.

THIROUX, A., & DUFOUGERÉ, W. (1910). Persistance de l'infection des méninges chez un singe guéri sans médication d'une infection sanguine à spirilles naturelle. *Ibid.*, III. 23.

THOMSON, J. G. (1923). The common occurrence of pseudo-spirochætes in the blood of blackwater fever cases, and of normal human beings and animals, when using the technique of Blanchard and Lefrou. *Journ. Trop. Med. & Hyg.*, XXVI, 251; *Brit. Med. Journ.*, I, 986.

TODD, J. L. (1920). *Spirochæta recurrentis :* a filter passer. *Journ. Parasit.*, VI, 152.

TODD, J. L., & WOLBACH, S. B. (1912). Parasitic Protozoa from the Gambia. *Journ. Med. Res.*, XXVI, 195.

UHLENHUTH & FROMME (1916). Untersuchungen über die Aetiologie, Immunität und spezifische Behandlung der Weilschen Krankheit (Icterus infectiosus). *Zeitschr. Imm.-Forsch.*, XXV, 317.

—— —— (1919). Experimentelle Untersuchungen über den Infektionsmodus, die Epidemiologie und Serumbehandlung der Weilschen Krankheit (Icterus infectiosus). II. Mitt. *Ibid.*. XXVIII, 1.

UHLENHUTH & ZUELZER (1921). Zur Epidemiologie der Weilschen Krankheit—zugleich ein Beitrag zur Frage der freilebenden Spirochäten (*Icterogenes*-ähnliche und andere). (8. Tag. d. Frei. Ver. f. Mikrob., Jena.) *Centralbl. Bakt.*, I. Abt., LXXXV (Beih.), 141*.

VELU, [H.] (1916). Sur la spirillose équine au Maroc. *Rec. Méd. Vét.*, XCII, 215.
VERVOORT, H. (1923). Spirochæten bij acute koortsige ziekten van onbekenden oorsprong in de tropen. Spirochætosis febrilis. *Geneesk. T'ijdschr. Nederl.-Indië*, LXIII, 800.
VINCENT, H. (1922). Sur la nature de la bronchite sanglante (fuso-spirochétose bronchique). *C. R. Soc. Biol.*, LXXXVI, 1002.
VUILLEMIN, P. (1905). Sur la dénomination de l'agent présumé de la syphilis. *C. R. Acad. Sci.*, CXL, 1567.

WARTHIN, A. S., BUFFINGTON. E., & WANSTROM, R. C. (1923). A study of rabbit spirochætosis *Journ. Infect. Dis.*, XXXII, 315.
WELLMAN, F. C. (1905). Case of relapsing fever, with remarks on its occurrence in the tropics and its relation to "tick fever." *Journ. Trop. Med. & Hyg.*, VIII, 97.
WENYON, C. M. (1906). Spirochætosis of mice due to "*Spirochæta muris*" n. sp. in the blood. *Journ. Hyg.*, VI, 580.
WERNER, H. (1909). Ueber Befunde von Darmspirochäten beim Menschen. *Centralbl. Bakt.*, I. Abt., LII, 241.
WHITE, W. C., & PRÖSCHER, F. (1907). Spirochætes in acute lymphatic leukemia and in chronic benign lymphatosis (Hodgkin's disease). Second communication. *Journ. Amer. Med. Assoc.*, XLIX, 1115.
WHITTINGHAM, H. E. (1921). Laboratory meeting. [Spirochætes in sand-fly fever.] *Trans. Roy. Soc. Trop. Med. & Hyg.*, XV, 149.
WILLIAMSON, G. A. (1908). Spirochætosis of Cypriote fowls. *Journ. Trop. Med. & Hyg.*, XI, 181.
WITTROCK, O. (1913). Beitrag zur Biologie der Spirochaeta des Rückfallfiebers. *Zeitschr. Hyg. u. Infekt.*, LXXIV, 55.
WOLBACH, S. B., & BINGER, C. A. L. (1914). Notes on a filtrable spirochæte from fresh water, *Spirochæta biflexa* (a new species). *Journ. Med. Res.*, XXX, 23.

YAMADA, K. (1917). Rat-bite fever due to the scratch of a cat. *Saikingaku Zasshi*, No. 265, 877; review *Trop. Dis. Bull.*, XIII, 340.
YORKE, W., & MACFIE, J. W. S. (1921). Laboratory meeting. *Trans. Roy. Soc. Trop. Med. & Hyg.*, XV, 149; *Accra Lab. Rept.*, 1921, 45.

ZUELZER, M. (1910). Ueber *Spirochæta plicatilis* und *Spirulina*. *Zool. Anz.*, XXXV, 795.
—— (1918). Beiträge zur Kenntnis der Morphologie und Entwicklung der Weilschen Spiro-chaete. *Arb. K. Gesundheitsamte*, LI, 159.
—— (1921). Biologische und systematische Spirochaetenuntersuchungen. (8. Tag. d. Frei. Ver. f. Mikrob. Jena.) *Centralbl. Bakt.*, I. Abt., LXXXV (Beih.), 154*.
—— (1922). Freilebende Wasserspirochaeten als Krankheitserreger. (9. Tag. d. Deutsch. Ver. f. Mikrob. Wurzburg.) *Ibid.*, LXXXIX, 171*.
—— (1925). Die Spirochaten. Prowazek's *Handb. d. Path. Prot.*, Lief. XI.
ZUELZER, M., & OBA, S. (1923). Beitrag zur Kenntnis saprophytischer Spirochaten. *Centralbl Bakt.*, I. Abt., XCI, 95.

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